



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

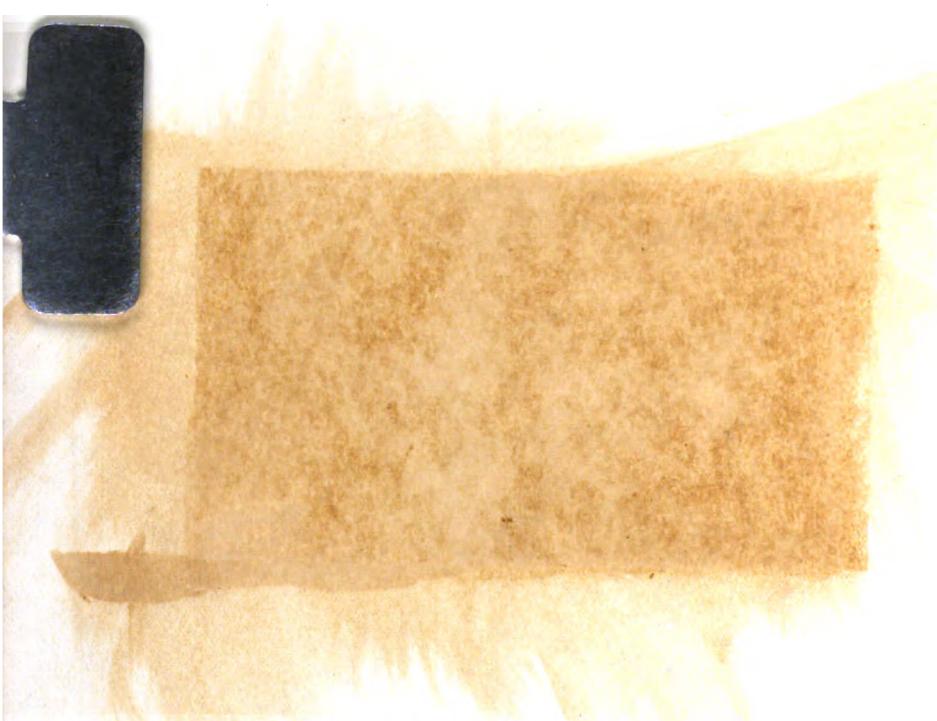
- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>



International clinics



610,5
16
c64

INTERNATIONAL CLINICS:

A QUARTERLY OF CLINICAL LECTURES

ON

MEDICINE, NEUROLOGY, SURGERY, GYNÆCOLOGY,
OBSTETRICS, OPHTHALMOLOGY,
LARYNGOLOGY, PHARYNGOLOGY, RHINOLOGY,
OTOTOLOGY, AND DERMATOLOGY,

AND SPECIALLY PREPARED ARTICLES ON TREATMENT AND DRUGS.

BY PROFESSORS AND LECTURERS IN THE LEADING
MEDICAL COLLEGES OF THE UNITED STATES,
GERMANY, AUSTRIA, FRANCE, GREAT
BRITAIN, AND CANADA.

EDITED BY

JUDSON DALAND, M.D. (UNIV. OF PENNA.), PHILADELPHIA,

Instructor in Clinical Medicine and Lecturer on Physical Diagnosis in the University of Pennsylvania;
Assistant Physician to the Hospital of the University of Pennsylvania; Professor of
Clinical Medicine in the Philadelphia Polyclinic; Fellow of the
College of Physicians of Philadelphia.

VOLUME III. NINTH SERIES. 1899.

PHILADELPHIA:

J. B. LIPPINCOTT COMPANY.

1899.

Copyright, 1899, by J. B. LIPPINCOTT COMPANY.

PRINTED BY J. B. LIPPINCOTT COMPANY, PHILADELPHIA, U.S.A.

CONTRIBUTORS TO VOLUME III. (NINTH SERIES.)

Baginsky, Professor Adolf, Director to the Kaiser und Kaiserin Friedrich Kinderspital (Children's Hospital), Berlin, and Professor of Diseases of Children at the University of Berlin, Germany.

Benedict, A. L., A.M., M.D., Professor of Psychology and Digestive Diseases, Dental Department, University of Buffalo; Special Consultant in Medicine, Buffalo Hospital of the Sisters of Charity, Buffalo, New York.

Bernhardt, Professor M., Professor of Nervous Diseases at the University of Berlin, Germany.

Bishop, Louis Faugères, A.M., M.D., Chairman Section on Medicine, New York Academy of Medicine; Attending Physician Colored Hospital; Assistant Physician French Hospital, etc., New York.

Cecil, John G., B. S., M.D., Professor of the Principles and Practice of Medicine and of Clinical Medicine in the Louisville Medical College, etc., Louisville, Kentucky.

Cumston, Charles Greene, B.M.S., M.D., Assistant Professor of Surgical Pathology, Faculty of Medicine, Tuft's College, Boston; Honorary Member of the Surgical Society of Belgium; Fellow of the American Association of Obstetricians and Gynaecologists; Corresponding Member of the Association of Genito-Urinary Surgeons of France, of the Obstetrical and Gynaecological Society of Paris, of the Pathological Society of Brussels, etc., Boston, Massachusetts.

Dabney, Samuel G., M.D., Professor of Psychology and Clinical Lecturer on Diseases of the Eye, Ear, Nose, and Throat in the Hospital College of Medicine, etc., Louisville, Kentucky.

Dieulafoy, Professor, Professor of Internal Pathology, Faculty of Medicine, Paris, France.

Eshner, Augustus A., M.D., Professor of Clinical Medicine in the Philadelphia Polyclinic; Physician to the Philadelphia Hospital, etc., Philadelphia, Pennsylvania.

Garré, Professor, Director of the Surgical Clinic and Professor of Surgery at the University of Rostock, Germany.

Gärtner, Professor G., Vienna, Austria.

Gerhardt, Professor C., Director of the Second Medical Clinic and Professor of Special Pathology and Therapy at the University of Berlin, Germany.

Gibson, William M., M.D., Utica, New York.

Goldscheider, Professor A., Medical Director of Moabit Hospital and Professor of Special Pathology and Therapy at the University of Berlin, Germany.

Grancher, Professor J., M.D., Professor of Clinical Medicine, Paris Faculty, France.

Hays, John E., A.M., M.D., Professor of Dermatology in the Hospital College of Medicine, etc., Louisville, Kentucky.

Helperich, Professor H., Professor of Surgery and Director of the Surgical Clinic at the University of Greifswald, Germany.

Hopkins, S. D., M.D., Associate Professor of Neurology in Gross Medical College, and Neurologist to the Arapahoe County Hospital and St. Anthony's Hospital, Denver, Colorado.

Hotz, F. C., M.D., Professor of Ophthalmology in the Chicago Polyclinic and of Ophthalmology and Otology in Rush Medical College, Chicago, Illinois.

Jackson, Edward, A.M., M.D., Denver, Colorado.

Krehl, Professor Ludwig, Director of the Polyclinic and Professor of Special Pathology and Therapy at the University of Jena.

Küster, Professor Ernst, Director of the Surgical Clinic and Professor of Surgery at the University of Marburg, Germany.

Lautenbach, Louis J., Jr., A.M., M.D., Ph.D., Surgeon in charge of the Philadelphia Eye, Ear, Nose, and Throat Institute; Nose and Throat Physician to the Odd-Fellows' Homes, etc., Philadelphia, Pennsylvania.

Leviseur Fred. J., M.D., New York.

Lloyd, James Hendrie, A.M., M.D., Neurologist to the Philadelphia Hospital, Philadelphia, Pennsylvania.

Marcy, Alexander, Jr., M.D., Riverton, New Jersey.

Mathews, Joseph M., M.D., LL.D., Professor of Surgery and Clinical Lecturer on Diseases of the Rectum in the Hospital College of Medicine, etc., Louisville, Kentucky.

Montgomery, E. E., M.D., Professor of Gynaecology in the Jefferson Medical College; Gynaecologist to Jefferson and St. Joseph's Hospitals; Ex-President of the Philadelphia Obstetrical and of the Pennsylvania State Medical Society, etc., Philadelphia, Pennsylvania.

Munro, John C., M.D., Assistant Visiting Surgeon, Boston City Hospital; Instructor in Surgery, Harvard Medical School, Boston, Massachusetts.

Plicque, A. F., M.D., Chief of Laboratory, Lariboisiere Hospital, Paris, France.

Robin, Albert, M.D., Assistant Professor at the Paris Faculty of Medicine, Paris, France.

Schultze, Professor Friedrich, Professor of Special Pathology and Therapy and Director of the Medical Clinic of the University of Bonn, Germany.

Stintzing, Professor R., Professor of Special Pathology and Therapy, and Director of the Medical Clinic at the University.

Strassman, Professor Fritz., Director of the Morgue and Professor of Legal Medicine at the University of Berlin, Germany.

Warner, Francis, M.D. (Lond.), F.R.C.P., F.R.C.S. (Eng.), Physician and Lecturer on Clinical Medicine and on Therapeutics in the London Hospital, England.

CONTENTS OF VOLUME III. (NINTH SERIES.)

Drugs and Remedial Agents.

	PAGE
ON SOME IMPROVEMENTS IN DAIRY TECHNICS AND THEIR MEDICAL IMPORTANCE. By PROFESSOR G. GÄRTNER	1
THE THERAPEUTIC VALUE OF EVAPORATORS IN THE LIV- ING ROOM. By LOUIS J. LAUTENBACH, A.M., M.D., PH.D.	7

Treatment.

TREATMENT OF INCIPIENT APPENDICITIS; MALIGNANT NEOPLASM OF THE VERTEBRÆ; CANCER OF THE CESOPHAGUS. By PROFESSOR R. STINTZING	15
THE TREATMENT OF TUBERCULOSIS. By PROFESSOR J. GRAN- CHER, M.D.	25
SURGICAL TREATMENT OF TUBERCULOUS NEPHRITIS. By PROFESSOR ERNST KÜSTER	38
THE DIAGNOSIS, MANAGEMENT, AND TREATMENT OF PLEURISY WITH EFFUSION. By LOUIS FAUGÈRES BISHOP, A.M., M.D.	48
THE TREATMENT OF RECENT AND OLD FRACTURES OF THE PATELLA. By PROFESSOR H. HELPERICH	50
THE TREATMENT OF TABES DORSALIS. By PROFESSOR A. GOLDSCHIEDER	60
TREATMENT OF CHOREA. By S. D. HOPKINS, M.D.	78
THE SURGICAL TREATMENT OF BENIGN AND MALIGNANT STRICTURES OF THE CESOPHAGUS. By PROFESSOR GARRE .	78
REMARKS UPON THE TREATMENT OF DIPHTHERIA, WITH ESPECIAL REFERENCE TO THE TECHNIQUE OF INTU- BATION. By SAMUEL G. DABNEY, M.D.	89
THE OPERATIVE TREATMENT OF HIGH MYOPIA. By EDWARD JACKSON, A.M., M.D.	97
THE TREATMENT OF FAVUS OF THE NAIL. By FRED. J. LEVISEUR, M.D.	105

Medicine.

	PAGE
DISTURBED CIRCULATION AND ITS EFFECTS ON THE BRAIN. By FRANCIS WARNER, M.D. (Lond.), F.R.C.P., F.R.C.S. (Eng.)	107
SYPHILIS OF THE KIDNEYS; SYPHILITIC NEPHRITIS; FORMS AND TREATMENT OF SYPHILIS OF THE KIDNEYS. By PROFESSOR DIEULAFOY and by A. F. PLICQUE, M.D.	116
CYCLIC VOMITING. By ALEXANDER MARCY, JR., M.D.	127
ANCHYLOSTOMIASIS AND GRAVES'S DISEASE. By PROFESSOR FRIEDRICH SCHULTZE	189
ACUTE FORMS OF GAS IN THE STOMACH; TWO TYPES AND A THEORY. By A. L. BENEDICT, A.M., M.D.	149
ON DIABETIC COMA AND ITS TREATMENT. By ALBERT ROBIN, M.D.	166
RUBEOLA; TYPHOID IN CHILDREN; SARCOMA OF THE LIVER. By PROFESSOR LUDWIG KREHL	170
THE BEARING OF EMBOLISM ON THE COURSE OF VALVULAR HEART-DISEASE. By WILLIAM M. GIBSON, M.D.	177
THE COMPLICATIONS OF CROUPOUS PNEUMONIA. By JOHN G. CECIL, B.S., M.D.	184

Neurology.

NEURASTHENIA. By AUGUSTUS A. ESHNER, M.D.	192
TRAUMATIC NEUROSIS AND A QUESTION OF DAMAGES. By PROFESSOR FRITZ STRASSMAN	199
OPTIC NEURITIS AS AN EARLY SYMPTOM IN NERVOUS DISEASES. By JAMES HENDRIE LLOYD, A.M., M.D.	209

Surgery.

GENERAL REMARKS UPON RECTAL DISEASES, WITH ESPECIAL REFERENCE TO THE TREATMENT OF FISTULA. By JOSEPH M. MATHEWS, M.D., LL.D.	216
BONE LESIONS OF HEREDITARY SYPHILIS IN CHILDREN. By PROFESSOR ADOLF BAGINSKY	224
TECHNIQUE OF HÆMOSTASIS IN OPERATIVE LESIONS OF THE LARGE VEINS. By CHARLES GREENE CUMSTON, B.M.S., M.D.	234
CASES OF ACUTE TRAUMATIC ABDOMINAL HEMORRHAGE. By JOHN C. MUNRO, M.D.	248

Gynaecology and Obstetrics.

	PAGE
APOPLEXY DURING PREGNANCY. By PROFESSOR C. GERHARDT	255
OVARIOTOMY. By E. E. MONTGOMERY, M.D.	265

Ophthalmology.

THE SIMULATION OF BLINDNESS AND ITS DETECTION. By F. C. HOTZ, M.D.	272
--	-----

Laryngology.

CLONIC SPASM OF THE SOFT PALATE WITH TICKING SOUNDS IN THE EAR; EPICONDYLALGIA. By PROFESSOR M. BERNHARDT	276
---	-----

Dermatology.

HERPES ZOSTER. By JOHN E. HAYS, A.M., M.D.	285
---	-----

LIST OF ILLUSTRATIONS TO VOLUME III. (NINTH SERIES.)

PLATES.

	PAGE
Photograph of four finger-nails affected by favus (Fig. 1), and a photograph of the same case cured (Fig. 2)	104
Photograph of finger-nails after removal from a case of favus (a) outside, and (b) inside (Fig. 3); photograph of nail of little finger undermined by favus (Fig. 4), and little finger of the same patient four weeks after removal of the nail (Fig. 5)	106
Photograph of the brain after hemorrhage (Fig. 1) and after strangulation (Fig. 2)	108

FIGURES.

Cut illustrating an evaporator suitable for the living room	11
Diagram illustrating the parallel bars and walking track with triangular obstacles to teach tabetics coördination and exact movements in walking (Fig. 1), and diagram showing step-ladder, on the rungs of which or between them a patient may put his feet while lying in bed, and so practise exact foot motions (Fig. 2)	71

Drugs and Remedial Agents.

ON SOME IMPROVEMENTS IN DAIRY TECHNICS AND THEIR MEDICAL IMPORTANCE.

LECTURE DELIVERED TO THE BERLIN SOCIETY FOR INTERNAL MEDICINE.

BY PROFESSOR G. GÄRTNER,
Of Vienna.

GENTLEMEN,—In his recently published book, “Outlines of the Nutrition of the Sick,” Professor Moritz says, “No experienced physician, I suppose, would be willing to dispense with any of the means at our disposal for the nutrition of the sick. . . . But if the question of their comparative value arose, I do not doubt that the first place would be allotted to milk.”

The procuring and preparation of milk and its products have made very great progress of late; a regular science of milk, with a great literature of monographs and special periodicals, has developed. The results of these researches are highly interesting to us, and deserve attentive consideration.

I will try to illustrate this by two very recent examples. You know that tuberculosis is spreading so rapidly among cattle that it is feared all cattle will ere long be tubercular. This circumstance has many evil consequences. Apart altogether from the facts that some animals die, and that the flesh of many becomes quite uneatable or of diminished value; quite apart, also, from the fact that the milk of many tubercular animals contains virulent tubercle-bacilli, the first consideration for the agriculturist is that such animals turn their food to less advantageous account than healthy ones, and that the enormous capital invested in herds of cattle yields much less interest. It

is therefore intelligible that the parties concerned are continually considering how this great evil may be remedied.

Bang pointed out very recently that the new-born calves are not tubercular, but that very many of them become so a few weeks after birth. From this he concluded, justly, I believe, that they are infected by the milk and perhaps by contact with the tongues of their dams. He therefore proposed that they should be at once removed from them, lodged in separate stalls, and fed with boiled milk. Such experiments were first made in Denmark, and are now frequently made here with the Prussian government's support. In this very simple and inexpensive way cattle free from tuberculosis have actually been obtained. We medical men may learn something from this. There are still enthusiasts who advocate the feeding of infants with unboiled milk,—that is, with milk which has proved dangerous for calves.

A second example, closely connected with the first, is the question whether butter can be a medium of infection. You know that a number of publications have appeared of late showing that virulent tubercle-bacilli are to be found in market butter. Other bacilli, which are very like tubercle-bacilli and have been confounded with them, have also been found in it. Whether the percentage of butter containing tubercle-bacilli is very great is still under discussion, but there is no doubt that a certain percentage of the specimens of market butter contain these bacilli in a virulent state. Now, though people may dispute as to whether it is very dangerous to eat such butter, it certainly is not quite harmless. If, now, we hear that dairy technics can easily produce butter absolutely free from living tubercle-bacilli by heating the cream up to 70° C. before buttering, we must say this, too, is a matter that interests us. I may add that butter has hitherto been made of heated (pasteurized) cream, in order to make it keep and bear transport better. Such butter can be bought, and we medical men, strictly speaking, ought to see that all butter, or, at least, all butter that is to be eaten raw, is pasteurized.

After these preliminary remarks I now address myself to the real theme of my lecture,—namely, the progress made in dairy technics in the direction of enabling us to alter the composition of milk at will.

Cow's milk is originally destined for the nourishment of calves, and is certainly admirably adapted to that purpose. It is not fitted, however, for the nourishment of infants, for they cannot digest it in

the first weeks of life. From of old, therefore, efforts have been made to render it more digestible, and this is done by dilution. Dilution diminishes the content of indigestible casein, and diluted milk does not curdle in such coarse flakes in the stomach as undiluted. This circumstance is probably of great importance. It is pretty generally accepted nowadays that the function of the stomach is to convert the food conveyed into it into a liquid or thin pulpy condition, and that the food remains so long in the stomach as this process lasts. It is proved by an investigation made by Dr. Schütz, of Vienna, that milk which curdles in fine flakes leaves the stomach much more quickly than that which curdles in coarse flakes.

Dilution, however, has one drawback. The nutritive value of cow's milk and human milk is nearly the same (the calorific value of both amounts to about sixty-two for one hundred cubic centimetres), but this value is of course diminished by dilution. In milk mixed with an equal quantity of water it is only thirty-one calories. By adding sugar of milk, indeed, it can be somewhat increased. But, as the calorific value of sugar of milk is much less than that of fat, it would be necessary, in order to effect a complete compensation, to add very much sugar of milk, which is impracticable for other reasons. The calorific value of sugared milk, therefore, is also less than that of human milk.

I have described a very simple process for the production of a milk whose casein content is reduced, while its fat content is so considerable that its calorific value is nearly equal to that of human milk. The process is effected by means of apparatuses that have long been known in dairy technics, and it was certainly due only to the circumstance that medical men have paid little attention to dairy technics, and dairy technicians little to the requirements of medical men, that it was reserved for me to devise this method, and thus in the simplest manner to solve a problem which had been solved till then in a very imperfect and very complicated way, and which many eminent specialists for the diseases of children had regarded as insolvable.

Let us suppose that we dilute one hundred litres of cow's milk with one hundred litres of water, thus reducing the casein content and the fat content by one-half. We then transfer this mixture to the milk-separator, or centrifuge, the main part of which is a drum, into which the milk flows, and which revolves very quickly. The

milk is driven towards the walls of this drum, and its light ingredients separate from its heavy ones, just as milk, when left quietly standing, separates in time into a layer of cream and a layer of skim-milk. The same thing happens here, the only difference being that in the centrifuge the process lasts just as many seconds as it lasts hours in standing milk. The drum has two outlets,—one in the periphery, through which the skim-milk, containing hardly any fat, flows off; the other reaching more into the middle of the drum, through which the fat milk flows. One can regulate the apparatus so that milk of quite definite fat content flows through the second outlet; milk, for instance, with only half the casein of the original milk and all the fat. I will add that one has it entirely in one's power to produce milk of any other composition one pleases, and in this way, in accordance with the proposal of Professor von Noorden, of Frankfort-on-the-Main, a milk is produced, which is first diluted with three parts of water. Its casein and sugar are thus reduced to one-quarter of their original amount, so that the sugar content is only one per cent. This diluted mixture is poured into the centrifuge again, which is so adjusted that milk of six per cent. fat content flows out of the cream-opening. The casein and sugar are not influenced by the centrifugal motion, but only the distribution of the emulsified fat is influenced. Such milk possesses a very high calorific value and a very small content of sugar, so that we can give it to diabetic patients in large quantities without misgiving; whereas, owing to its large sugar content, ordinary milk can be given to such patients only in small quantities.

My method is extremely simple, and the medical men who visit the recently opened establishment "Schweizerhof" will be able to convince themselves by personal inspection that a few minutes, or rather seconds, suffice for the conversion of a quantity of milk from one state into the other. Unlike other recently devised methods of preparing milk for infants, the process is not a complicated chemical but a *simple mechanical* one. Heubner's very just demand that the milk's way from the cow to the child shall not be lengthened is certainly complied with in full measure.

The preparation of fat-milk (this name was given the new milk by Professor Escherich) consists, as already stated, of only two processes. The first is dilution, but milk intended for infants has always been diluted, so this cannot be laid to my charge as a complication.

The second process is the centrifugal treatment, and it has been objected by many that this impairs the emulsion state of the milk. It is natural to suppose that the milk is tremendously shaken about in the centrifugal drum, but that is by no means the case. When it flows in it lays itself against the side of the drum; then it rotates with the centrifuge, and the particles shift but little in this process. Only, the fat moves inward quicker than when the milk is allowed to stand. A shaking of the milk, such as takes place in buttering, is out of the question.

By my process nearly the same purpose is gained as if cattle-breeders had succeeded in producing a race of cattle whose milk had 1.5 per cent. of casein and 3.2 per cent. of fat, which milk we should certainly regard as very well adapted for the nutrition of infants.

Of the results that have been obtained with fat-milk I will say no more. There is a very large amount of literature on the subject. A considerable number of writers have expressed approval; others, and especially those who have tried it with sick children, were less satisfied. One thing is certain: there is no danger in the matter, and I may therefore beg you to make experiments yourselves and convince yourselves personally whether fat-milk is better than that hitherto given to infants or not.

I have still a few words to say regarding the use of fat-milk in other cases. It is often given with admirable success to persons suffering from stomachic complaints. It is generally known that ordinary milk often does not agree with such patients, especially with those who suffer from *ulcus rotundum*. Experience has shown that fat-milk frequently agrees with them, evidently because it curdles in much finer flakes than unskimmed milk. It must be remembered that the nutritive value of both is the same. At Carlsbad fat-milk is used instead of ordinary milk by those who take the waters, and is drunk in large quantities, especially by patients suffering from *ulcus*. Dr. Laquer, of Wiesbaden, has tried fat-milk in many cases of gout, and declares that the results were good.

Fat-milk and, especially, diabetes milk differ somewhat in taste from ordinary unskimmed milk. The taste of milk depends first and foremost on its content of salt. Two kinds of milk, differing only in fat content, can hardly be distinguished by the palate. Nor does the sugar content play an important part. A reduction of the salt content, on the other hand, is noticed at once; it betrays itself by a

"watery" taste. Human milk, too, with its small salt content, is said to have an insipid, watery taste. It is natural to suppose that the taste might be corrected by the addition of salt. This cannot be done, however, with common salt, for it gives the milk a distinctly salty taste. But if one adds to each litre (two pints) from two to four grammes (one-half to one drachm) of a salt which has approximately the composition of milk-ashes (but without phosphate of lime), the taste of fat-milk or of diabetes milk becomes quite like that of unskimmed milk.¹

A few words more about another alteration of the taste of milk. The aversion of patients to the taste of *boiled* milk often frustrates all attempts to use it as an article of nourishment. If one heats milk in an open pot, and the temperature rises to 70° C., one notices a peculiar and disagreeable burnt taste and smell. I have found a method of boiling milk so that this taste is hardly, if at all, noticeable. One must prevent the contact of the hot milk with the air. This can be done in a very simple and convenient manner by pouring the milk into boiling-down glasses with cylindrical necks, and throwing into each glass a piece of pure paraffin as large as a walnut, which melts at 60°. These glasses are heated in a steam-cooking apparatus. The melting paraffin forms a connected layer of oil on the surface. One can boil the milk as long as one likes; the gases and vapors escape without the milk ever coming into contact with the air. When one takes the pot from the fire, the paraffin congeals on the surface, and forms a solid protecting cover, so that the milk may be kept for days, as if it were sterilized. The burnt taste is so slight that many people have thought the milk was unboiled. This experience may sometimes be very useful. Milk sterilized in streaming steam, like that prepared at the Schweizerhof according to the method of Neupan, Gronwald, and Oehlmann, has a much slighter taste of burning than that which comes into contact with air in boiling or after boiling.

¹ Salt is added only at special request, and only to milk intended for adults. Several times it has proved useful in the case of infants too. Children who would not take ordinary fat-milk drank it willingly when salt was added. The salt had also a good effect against existing constipation. The addition of salt would also meet a theoretic demand very recently put forward by P. H. Koeppe; it would make the osmotic pressure of fat-milk equal to that of human milk and of the blood. The justice of this demand can be proved only by practical experiments.

THE THERAPEUTIC VALUE OF EVAPORATORS IN THE LIVING ROOM.

READ BEFORE THE SECTION ON MATERIA MEDICA AND THERAPEUTICS AT THE
FIFTIETH MEETING OF THE AMERICAN MEDICAL ASSOCIATION, HELD AT
COLUMBUS, OHIO, JUNE 6 TO 9, 1889.

BY LOUIS J. LAUTENBACH, A.M., M.D., Ph.D.,

Surgeon in Charge of the Philadelphia Eye, Ear, Nose, and Throat Institute; Nose
and Throat Physician to the Odd-Fellows' Homes, etc., Philadelphia, Pa.

GENTLEMEN,—The need of a proper degree of atmospheric humidity has been recognized, but so little stress has been laid upon it that little or no exertion is usually made to provide for a deficiency in the living or even in the sick-room. Indeed, in many of our larger institutions, such as hospitals and asylums, the construction of the buildings indicates a woful lack of any special efforts having been made to provide for a supply of properly moistened air.

Years ago, in the days of stoves especially, it was rather a common story to have vessels of water scattered about the house in convenient places, with the view of restoring to the indoor air a little of the life which had been burned out of it by the modern need of heating apparatus. Naturally, these water-pitchers and basins were usually placed on the stove or immediately adjoining it, if the former was impossible. In their way, these vessels served a very useful purpose, both from a hygienic and therapeutic stand-point. Although this method was a crude one, it was in many respects the superior to the more modern ones, or rather want thereof, found in our homes of to-day.

As from fifty-eight to sixty-seven per cent. of the human body is water, it must necessarily follow that it must be liberally supplied with this constituent to make up for the loss which is so constantly occurring during all life's processes. It passes as perspiration from the skin, as vapor from the lungs, and is thrown off from all the excretory organs as well as from all mucous surfaces. To supply all this waste needs constant and frequent renewals, both by the mouth and by the skin of the body, as well as by the exposed mucous surfaces.

To supply these latter demands properly, and to allow comfort to the lungs, nose, throat, eyes, and to the entire body surfaces, it has been found necessary that the humidity of the atmosphere shall amount to about seventy-five per cent. of its saturation. If it fall much below this, it creates considerable discomfort,—the dry, east spring winds of England serve to illustrate this,—whereas, if it is near the saturation point, the oppression and sometimes almost utter inability to breathe properly are very noticeable; the inhabitants of our eastern seaboard often experience this in the early summer and fall, especially in the months of August and October.

Now, the saturation point of our atmosphere varies with the temperature, which will be observed by looking over the following table:

Air can absorb at	82° F.	the 160th part of its own weight of watery vapor.
" "	" 59° F.	" 80th "
" "	" 86° F.	" 40th "
" "	" 118° F.	" 20th "

For every rise in temperature of 27° the capacity of the air for water is doubled. This absolute humidity is rarely attained, never indoors, and would be as harmful as its opposite, no humidity. It occurs in tropical countries during the rainy season. Any degree of humidity beneath this is termed relative humidity, and, as before indicated, 75° is considered normal for man's atmosphere.

The quantity of water in each cubic foot of air varies greatly according to the temperature. This table, giving the actual weight of water contained in a cubic foot of air at different temperatures, may serve to illustrate that there is a wide variation in that necessary for the system, at different degrees of heat. Of course, it is to be remembered that three-fourths of the amount expressed in this table is to be considered normal:

AQUEOUS VAPOR IN A CUBIC FOOT OF SATURATED AIR AT DIFFERENT TEMPERATURES.

Degrees Fahrenheit.	0° F.	1° F.	2° F.	3° F.	4° F.	5° F.	6° F.	7° F.	8° F.	9° F.
heit.	Grains.									
0°	0.545	0.569	0.595	0.621	0.649	0.678	0.708	0.739	0.772	0.806
10°	0.841	0.878	0.916	0.957	0.999	1.043	1.090	1.138	1.190	1.284
20°	1.298	1.355	1.415	1.476	1.540	1.606	1.674	1.745	1.817	1.892
30°	1.969	2.046	2.126	2.208	2.292	2.379	2.469	2.563	2.659	2.759
40°	2.862	2.967	3.076	3.180	3.306	3.426	3.550	3.679	3.811	3.948
50°	4.089	4.234	4.383	4.537	4.696	4.860	5.028	5.202	5.381	5.562
60°	5.756	5.952	6.154	6.361	6.575	6.795	7.021	7.253	7.496	7.739
70°	7.992	8.252	8.521	8.797	9.061	9.372	9.670	9.977	10.292	10.616
80°	10.949	11.291	11.643	12.095	12.376	12.756	13.116	13.546	13.957	14.378
90°	14.810	15.254	15.709	16.176	16.651	17.145	17.618	18.164	18.698	19.285
100°	19.790	20.357	20.938	21.535	22.145	22.771	23.411	24.069	24.742	25.429

Now, if it is necessary to retain the humidity at seventy-five per cent. of saturation, we need some instrument to indicate the saturation point; the wet and dry bulb hygrometer of August is perhaps the most simple and, on the whole, the most satisfactory. It really consists of two thermometers, one of which is used as usual and the other has its bulb constantly saturated by trickling water on a cloth, one end of which is inserted in a vessel of water, the other being wrapped about the bulb. Now, by comparing these two thermometers, a difference of temperature will be observed, and by using the following table, which is condensed from Glashier's large table, the relative humidity is obtained at once, and the humidity can then be increased or decreased at will, if necessary, by suitable appliances.

The modern house is almost invariably too dry as soon as the windows need to be closed and the fires started,—the cause of this is several fold. If heated by the hot-air furnace, the air brought into the homes at such times is usually 32° F. or less, and by referring to the table you will observe that the amount of water contained in a cubic foot then amounts to about two and one-eighth grains. Now this air, brought into the cold-air flue, is heated by circulating about a heated cylinder, and an effort is usually made to add to its water vapor by bringing the air into contact with the surface of a small vessel of water rarely presenting as much as a square foot of water surface, and the air passing rapidly over this can indeed absorb *very* little of it. You will observe that at the saturation point the air, being heated to 70°, would contain almost eight grains of water vapor, almost four times as much as at 32°. In other words, it should absorb about four grains of water for each cubic foot of air entering the flues.

If this water were boiling, it would the more readily enter the atmosphere, but for the air in its rapid passage over such a small surface of water to increase its amount almost fourfold is impossible; the consequence is that, as it enters the room, it feels dry, burned out, and overheated. If the house be heated by steam or water radiators, it is true that the air has not the burned, dry feeling which is really due to the disproportionately low relative humidity in the heating of the air from 32° to 70° without a corresponding addition to its watery vapor, but by hygrometric testing the humidity will usually be found below fifty per cent.

If the house be heated by hot air passing over steam-pipes, there is usually but an attempt to add humidity to the air, and in conse-

TABLE OF THE RELATIVE HUMIDITY GIVEN BY THE DIFFERENCE BETWEEN THE DRY AND WET BULB.

Temperature of the Dry Bulb.	DIFFERENCE BETWEEN THE DRY AND WET BULB.															
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
	Relative Humidity; Saturation, 100.															
90	100	95	90	85	81	77	73	69	65	62	59	56	53	50	47	44
89	100	95	90	85	81	77	73	69	65	61	58	55	52	49	46	43
88	100	95	90	85	81	77	73	69	65	61	58	55	52	49	46	43
87	100	95	90	85	81	77	73	69	65	61	58	55	52	49	46	43
86	100	95	90	85	80	76	72	68	64	61	58	55	52	49	46	43
85	100	95	90	85	80	76	72	68	64	61	58	55	52	49	46	43
84	100	95	90	85	80	76	72	68	64	60	57	54	51	48	45	43
83	100	95	90	85	80	76	72	68	64	60	57	54	51	48	45	42
82	100	95	90	85	80	76	72	68	64	60	57	54	51	48	45	42
81	100	95	90	85	80	76	72	68	64	60	56	53	50	47	44	41
80	100	95	90	85	80	75	71	67	63	59	56	53	50	47	44	41
79	100	95	90	85	80	75	71	67	63	59	56	53	50	47	44	41
78	100	94	89	84	79	75	71	67	63	59	56	53	50	47	44	41
77	100	94	89	84	79	75	71	67	63	59	56	53	50	47	44	41
76	100	94	89	84	79	75	71	67	63	59	55	52	49	46	43	40
75	100	94	89	84	79	74	70	66	62	58	55	52	49	46	43	40
74	100	94	89	84	79	74	70	66	62	58	55	52	48	45	43	40
73	100	94	89	84	79	74	70	66	62	58	54	51	48	45	42	40
72	100	94	89	84	79	74	69	65	61	57	54	51	48	45	42	39
71	100	94	88	83	78	73	69	65	61	57	53	50	47	44	41	38
70	100	94	88	83	78	73	69	65	61	57	53	50	47	44	41	38
69	100	94	88	83	78	73	68	64	60	56	53	50	47	44	41	38
68	100	94	88	83	78	73	68	64	60	56	52	49	46	43	40	37
67	100	94	88	83	78	73	68	64	60	56	52	49	46	43	40	37
66	100	94	88	83	78	73	68	64	60	56	52	49	45	42	40	37
65	100	94	88	83	78	73	68	63	59	55	51	48	45	42	39	36
64	100	94	88	82	77	72	67	63	59	55	51	48	45	42	39	36
63	100	94	88	82	77	72	67	63	59	55	51	47	44	41	38	35
62	100	94	88	82	77	72	67	62	58	55	50	47	44	41	38	35
61	100	94	88	82	77	72	67	62	58	54	50	47	44	41	38	35
60	100	94	88	82	76	71	66	62	58	54	50	46	43	40	37	34
59	100	94	88	82	76	71	66	61	57	53	49	46	43	40	37	34
58	100	98	87	81	76	71	66	61	57	53	49	46	43	40	37	34
57	100	98	87	81	75	70	65	61	57	53	49	45	42	39	36	33
56	100	98	87	81	75	70	65	60	56	52	48	44	41	38	35	32
55	100	98	87	81	75	70	65	60	56	52	48	44	41	38	35	32
54	100	98	86	80	74	69	64	59	55	51	47	43	40	37	34	31
53	100	98	86	80	74	69	64	59	55	51	47	43	40	37	34	30
52	100	98	86	80	74	69	64	59	54	50	46	42	39	36	33	30
51	100	98	86	80	74	68	63	58	54	50	46	42	38	35	32	29
50	100	98	86	80	74	68	63	58	53	49	45	41	37	34	31	29
49	100	98	86	79	73	67	62	57	53	49	45	41	37	34	31	28
48	100	98	86	79	73	67	62	57	52	48	44	40	36	33	30	
47	100	98	86	79	73	67	61	56	51	47	43	39	36	33	30	
46	100	98	86	79	73	67	61	56	51	47	43	39	35	32	29	
45	100	92	85	78	72	66	60	55	50	46	42	38	34	31	28	
44	100	92	84	78	71	65	59	54	49	45	41	37	34	31	28	
43	100	92	84	78	71	65	59	54	49	45	41	37	34	31	28	
42	100	92	84	78	71	64	59	54	49	44	40	36	34	30	27	
41	100	92	84	77	70	64	58	53	48	43	39	35	31	28		
40	100	92	84	77	69	68	57	51	46	42	38	34	31	28		
39	100	92	84	77	69	63	57	52	47	42	38	34	31	28		
38	100	91	83	75	68	62	56	50	45	41	37					
37	100	91	83	75	68	61	55	49	44	39						
36	100	91	82	74	66	59	53	47	42							
35	100	90	80	72												
34	100	89	79	72												
33	100	89	78	70												
32	100	87	75													

quence the air here will suffer just as in the hot-air furnace; but not as much so, as it is usually heated to a lower temperature. This method is being adopted in many of our large institutions, schools, asylums, etc., and is quite faulty, unless provided with immense heated evaporating pans.

The method which I wish here to propose is one which I have used and advised for years. It consists in attaching to each radiator or register an earthen, terra-cotta, porous cylinder, of a capacity of about half a gallon. It should be constantly kept filled with water. In some houses it may be necessary to replenish it three or four times a day, and it should be cleaned frequently to prevent closure of the pores.



This cylinder here illustrated, ten inches high and four inches across, has a little projection or off-set, three and a half inches from the top, to allow of its being readily fastened to the register or radiator by means of a double hooked wire, as here illustrated, or by means of a cord or piece of ribbon. It is readily attached and as readily detached. The latter may be necessary occasionally to allow of its thorough cleansing. It is made of a terra-cotta material, so porous that much of the water percolates through the interstices, evaporating from the surface of the cylinder,—in other words, instead of evapo-

rating from the top water surface only, an area of about ten square inches, it evaporates from the entire sides, one hundred and twenty-five square inches, and base as well, twenty-five square inches, making a total of one hundred and fifty instead of ten square inches. Being in contact with the heat-flue, the water is heated and evaporates yet more readily than it would do otherwise. In consequence, the air is more thoroughly charged with water vapor by this method than by any of which I have any knowledge; the heat of the room is rendered more bearable through its means, equalizing its temperature, and the cold is not observed as much, as the water vapor present prevents rapid loss of body heat. Then again, the heat of the room is far more apt to remain constant, if there is a fair amount of water vapor present than if present in small percentage.

If the atmosphere surrounding us has in it less than the normal amount of water, then must the skin, the mucous linings of the lung-tubes, the nasal and pharyngeal mucous surfaces, as well as that of the eye, either suffer a degree of dryness leading to increased friction during functionation followed by local or later general febrility, or the deficiency must be made up by an increased activity of the local capillaries or nutrients causing a hyperæmia. That for a short time such a transference of work may overcome the difficulty is true; if long continued or occurring frequently, the local structures suffer from the condition, and diseased processes are induced which may develop into most serious conditions. This is especially prone to occur in the chest as well as in the nose and throat, although even in the eyes we have many conjunctival congestions and inflammations thus occasioned. Here the dry conjunctiva must have more secretion, as the external atmosphere, instead of supplying moisture to the eyes, is stealing it therefrom constantly, and this dryness is compensated for, first, by an increased activity of the tear-glands, and, secondly, an increase in the diameter and tortuosity of the local conjunctival vessels. These vessels enlarge, first, because of the extra need for a supply of moisture to the conjunctiva, and also because of the reflex from the irritability from rubbing of the dry conjunctiva over the dry cornea.

In the lungs this extremely dry air operates in the same way, by inducing a drying and adherence of the natural mucous discharges and a secondary enlargement of the ultimate vessels inducing a tendency to a breaking down of tissue. In the throat and nose the

effects are observable by a hard, irritable huskiness in the throat and larynx, an inability to swallow comfortably, with a diminution of the salivary secretion, and a consequent interference with digestion. In the nose we have, as a result, dryness with scabbiness and irritability, with increased friction in breathing and less of a cleansing of the air by the nose, as there is not sufficient moisture on its surface to hold the foreign particles. Many an atrophic nasal catarrh has, I think, been thus induced.

The general symptoms of excess of humidity and of a deficiency thereof have been too often experienced by each of us to need more than mention. The unaccountable general human pathological feelings, which often hold hundreds and thousands and sometimes, perhaps, millions within its grasp, are usually atmospheric, and often, no doubt, connected with an imperfect degree of humidity; and if we are able within our homes to regulate this and enjoy the therapeutic effects of this regulation, we will succeed, no doubt, in passing some of our hours in a more healthy condition and in a more enjoyable manner.

In the cases of pathological conditions of the head and chest thus induced, if we recognize the condition and rectify it, we will have good results very quickly, unless the conditions have existed too long or been repeated too often.

In many cases of corneal and conjunctival disease I pay strict attention to the humidity of the patient's apartments, and have found it make a material change for the better by using my evaporators throughout the house; especially noticeable have been the good effects brought about in old people, in whom naturally some of the watery body structure had been lost during life.

In nose and throat diseases, especially of acute types accompanied by pain and irritability, has the use of these porous pots been followed by good results. I mean by this that the good results of the ordinary treatment were observed more rapidly, or that, on the commencement of the natural humidity treatment, there was at once an additional change for the better, noticeable to the patient, the nurse, and the medical attendant.

In cases of tonsillitis, and of diphtheria especially, have I remarked a very happy effect on this addition, inasmuch as the patients seem at once to be more comfortable and restful, the pain moderating. It has the additional advantage that the bacilli in an atmosphere properly

charged with water vapor are less apt to peregrinate and develop on new soil.

In cases of consumptives, I have known of its use with advantage, apparently giving them a better breathing capacity, from the fact that the air supplied is more in accordance with the needs of the tissues, and, in addition, it ties the tubercle bacillus more firmly to the infected patient.

I do not claim that there is anything new and startling in this prevention and therapeusis, but I do claim that the question of relative humidity of the atmosphere is a very important one, which has received but little attention; that if well studied and the principles properly applied the human race would develop more perfectly and comfortably and rationally; that vague, uncertain conditions of the mind and body, which now affect people over wide areas of country, would be lessened in their effects; that not only would inflammatory diseases of the eye, nose and throat, and lungs be less frequent, but would be less fatal, and when they did occur, would be more amenable to treatment, and that this simple little porous pot will, time after time, indicate its presence by a very prompt change in the appearance of pathological conditions and upon mucous surfaces as well as a rapid amelioration of the symptoms.

The apparatus is inexpensive. It was made for me by Galloway & Co., and is now being sold by Emil Jungmann, of Philadelphia.

Treatment.

TREATMENT OF INCIPIENT APPENDICITIS; MALIGNANT NEOPLASM OF THE VERTEBRAE; CANCER OF THE OESOPHAGUS.¹

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF JENA.

BY PROFESSOR R. STINTZING,

Professor of Special Pathology and Therapy, and Director of the Medical Clinic at the University.

GENTLEMEN,—I pass around here the stool of the patient suffering from perityphlitis or appendicitis, whose case we discussed some days ago. You see that it is plentiful, of good color, and excellent consistency, a long-drawn-out, salvy stool. It was passed yesterday evening without the slightest inconvenience, and has all the characters of a normal stool. Our patient is under the opiate treatment, which, as you know, we consider the only rational internal treatment under the circumstances. He has not had a stool before in eleven days, and we have not considered it necessary to give him a purgative or take any means to secure an evacuation, as we felt sure that nature herself would take the best and most natural means to clear the fecal matter from a practically normal large intestine as soon as we had ceased quieting peristaltic movements by opiates.

Had an evacuation not occurred in four or five days more, then I would have thought of prescribing for him a mild purgative, but, under the circumstances, when he is on a limited diet, and that of the most digestible and completely absorbable material possible, I should consider it imprudently hasty if I were to force a bowel movement before fourteen days were past. You have in the present case an ex-

¹ Reported by James J. Walsh, Ph.D., M.D.

cellent exemplification of the expectant method in the treatment of appendicitis.

Our patient's temperature has gone down, his pulse has become practically normal, and the attack is past. There still remains some tenderness in the right iliac region; there is even an additional sense of resistance there, which I should consider due to a slight spasm of the cæcum intended to keep the parts absolutely at rest, rather than to the collection of exudate. Nature herself would thus point out *the* indication for treatment of the affection,—absolute rest of the parts involved in the inflammatory process,—and artificially we can best produce this state of physiological rest by the judicious use of the opiates.

I know that in this opinion I am thoroughly in accord with most of the surgeons, who claim that all of the cases of even suspected appendicitis belong by right to them, and who insist that the administration of opium, by masking the symptoms, obscures the course of the disease and makes it even harder to judge than it normally is (and it is always hard enough) when surgical intervention is demanded. They are apt to recommend purgatives, than which, to my mind, nothing is more contraindicated.

The bowel irritated by an inflammatory process is aroused to a state of active peristalsis, which in most cases becomes spasmotic and is the cause of a good deal of pain. This makes the patient restless and uneasy, lowers his nervous tone, and so makes him less than normally resistant to depressive influences. Nature proceeds to wall off the surrounding delicate structures from the risk of infection by an inflammatory exudate, the spasmotic condition and the pain serving, when not exaggerated, to keep the intestines at rest during this exudative process. To purge, as is so often advised, is to work directly against nature, to still further irritate an inflamed part of the bowel, to set up a state of peristaltic unrest, which prevents the possibility of the proper formation of adhesions and greatly increases the danger should infection of the peritoneum take place, either through the inflamed intestine or by a perforation of the appendix vermiformis.

The surgeons, who claim that nearly every case of appendicitis needs surgical treatment, point to a set of statistics that do not accord by any means with the experience of the physician. Certainly considerably more than the half of the cases of perityphlitis that we see in our medical wards run a mild and absolutely favorable course to

speedy and complete recovery. We have the opportunity to see a great many more such conditions than the surgeons to whom, as a rule, only the serious cases go.

Do not then be led astray by the false notion that the offending material which has set up the inflammatory process is still in the intestine and must be gotten out as soon as possible. Whatever proportion of cases must eventually come into the surgeon's hands may not be clear, but one thing in these perityphlitic cases seems beyond cavil, and that is that purgatives and, in general, efforts to clear out the intestine can only do harm. Opium is indicated for the pain and restlessness, and should be given freely though judiciously. Only enough should be given to relieve the pain and keep the patient in a state of ease. After days of waiting, when, as in our case here, a healthy stool has come, care must be enjoined on the patient, and he must not be allowed to get up too soon. Where there are evidences of an inflammatory exudate still at hand, then rest and the opium treatment must be continued. It may take some weeks, even longer at times, before all the local symptoms have disappeared, and occasional doses of opium, that will keep peristalsis quiet, will assist the natural process of absorption of the exudate. Anxiety about the patient's stools need not be too great, and if a movement is secured every eleven to fourteen days that will suffice during the continuance of the liquid, absolutely bland diet which is all the patient must be allowed.

You will be better satisfied with the results of a series of cases under this treatment than if you invariably refer your cases of suspected appendicitis to the surgeon. After all, even the best sets of surgical statistics show many a failure. Of course, there are cases where the care of a surgeon is evidently needed at the very beginning, cases of intense acuity, with high fever and great pain that is localized to a particular spot; other cases, too, where the occurrence of fluctuation points to the necessity of evacuating a collection of pus. Then there are a series of cases of perityphlitis, most frequently appendicial in origin, that are most insidious in their origin and may be very serious in their outcome, where the temperature mounts for several days, or where there are considerable variations of temperature, or, finally, where the local or general symptoms do not remit despite the quieting effects of the opiates. Such cases will always be a matter for the most serious consideration. In them the armed ex-

pectancy of the surgeon finds its true place, and you must always have a surgical colleague; but this must not let you be carried away by the *furor operandi*, which certainly does at least as much harm as good.

Our first patient, gentlemen, is a case for which we can do very little except to still pain and lessen the torments of his painful affection by the administration of opiates. He is extremely interesting, however, from a diagnostic stand-point, and I wished you to see him, as the opportunity to study such cases does not come often. He is, as you see, in a most pitiful state, constantly moaning and complaining, though his senses are so benumbed by morphine that it requires considerable effort to arouse him to have him answer a question. When asked if he is in pain, he says, "Oh, yes! Oh, yes! Oh, yes!" When we ask him, Where? he seems to have lapsed again into obliviousness of his surroundings, until again aroused and the question repeated, when he says in his back, and makes a movement as if to indicate where.

He is evidently in great discomfort, though, as you may judge from even these answers, his sensorium is reasonably free and his mind clear. His illness began about two months ago with paraparesis of his legs, weakness which soon became a paraplegia, and has been followed by contractures. You notice, here, his knees are bent at an acute angle, and his heels are drawn up until they touch his buttocks. When we attempt to stretch them out we meet with almost insuperable resistance, and movement is followed by renewed complaint of pain and discomfort.

In his present state we can get nothing of his history from him, but some weeks ago, from himself and his friends, we succeeded in gathering this much of his history, which is almost necessarily fragmentary: He is about forty-five years of age; is a peasant living in the country; his mother died of tuberculosis; he is married, and his wife and children are living and well. About two months ago, as we have said, he noticed weakness in his legs; this was soon followed by paraplegia that confined him to bed and gave the chance for these contractures to form. Bladder and rectal disturbances soon developed, and, besides the motor palsy, anaesthesia began to develop in his legs. Meantime, however, there had been shooting pains in his limbs and girdle sensations in his trunk. Then pain became the main feature of the affection, until he lay all day and all night in constant

agony from pains in all the lower part of his body, with a painful sense of pressure all over. All this developed in about four weeks, when he was brought to us. We have been able only to make life bearable for him to a certain degree by the constant administration of opiates.

What is of special interest in the case is the diagnosis and the prognosis that depend on it. There is evidently a lesion of the cord. The paraplegia, the contractures, the bladder and rectal disturbances, and the hypæsthesia might make us think of chronic myelitis, but there is no history of an infectious disease preceding the first symptoms, and while the course of the disease is most too slow for acute myelitis, it is too rapid and pain is too prominent a symptom for chronic myelitis. We have no reason to think of syphilis in the case; he has no scars of it on his body, and his wife and children are healthy.

The suspicious circumstance that his mother died of tuberculosis of the lungs adds weight to the opinion that we may have to do with a tubercular lesion either of the vertebræ or the meninges. In most cases where paraplegia develops without an obvious cause, it is well to think of a tubercular degeneration of the vertebræ,—so-called Pott's disease. We do not find, however, any projecting prominent point along the spine, and while one part here of the lower dorsal region is perhaps tenderer than others, his benumbed condition and his constant complaints make it very hard to say whether there is really a tenderness from a definite anatomical lesion at this point.

Our patient is older than patients usually are who develop tuberculosis of the vertebræ, as it is especially a disease of the developmental period, and only rarely begins after twenty-five. Besides, the symptoms have come on too rapidly and the pain is too severe. This rapid course and excruciating continuous pain are very characteristic of malignant neoplasm of the vertebræ. The posterior roots are pressed upon by the rapidly growing neoplasm, and the result is neuralgic pains of the most terrible description. They are sometimes conducted along the nerve-trunks to the extremities, and put the patient in the intensely pitiful condition that you see here. The slightest movement of the extremities causes agony, and a state of intense cutaneous hyperæsthesia is developed, in which the slightest touch is painful.

Primary malignant neoplasm of the vertebræ is, however, com-

paratively very rare, yet we have no symptoms that point to the existence of a primary neoplasm in any of the internal organs. There is no tumor in the region of the stomach, none in the rectum, and no nodules are to be found in the breasts or the prostate, for it is from these latter two organs especially that metastases to the vertebræ are most frequent. Just because our patient happens to be a male is no reason why we should neglect to examine his mammary glands for the presence of the primary neoplasm, for carcinoma of the male breast is not rare enough to make that procedure unnecessary or absurd. While the growth in the vertebræ may be primary, it is probable that the autopsy will disclose a small nodule of cancer in one of the internal organs, probably the stomach, from which it has originated.

At his age it is probably cancer, not sarcoma, that we have to do with. It is probable that, unfortunately, or perhaps fortunately for our patient in his suffering condition, we shall not have to wait long for the autopsy to decide our questions. His symptoms have grown much worse in these last few days, and his present condition is an index of approaching dissolution not hard to make out.

For therapy we have only the symptomatic treatment of his pitiable condition by opiates. In such cases, despite the seemingly complete exclusion of syphilis in his personal and family history, it is always well to try the effects of mercurial inunctions and the iodides. Here they have had no effect, but we shall continue their use, for while there is life there is some therapeutic hope.

Our next patient, gentlemen, is a man of forty-six, who since last autumn has lost in weight and who has felt that he was losing strength. You can see how emaciated he is now, though a good deal of that is due to inanition in the last few weeks. About two weeks ago, after eating some spinach, as he tells us, he had a sudden regurgitation from the mouth and nose, and since then has been able to swallow but very little. This brought him to the hospital, where we got briefly the following history:

He is a workman in a brewery, and his personal and family history presents no points of interest for us. For some time he has had gradually increasing difficulty in swallowing. At first he had to be careful to chew his food very fine, or it did not go down at all, but was regurgitated. Then he could take nothing but liquid food. Since his coming to the hospital we have found that even liquid food

does not, after being swallowed, always find its way into the stomach, but that, even when taken in but small quantities, it is after a time regurgitated. I use the word regurgitated advisedly, because it is not vomited, in the proper sense of the word, since it has never been in the stomach.

I pass some of the material around that he has brought up this morning, though he has had but a little coffee, absolutely nothing else, before being brought to the clinic. There is a brownish-colored froth on top of it that is coffee-colored, from his morning drink. The fluid itself is mainly mucus, though there is a layer of white fluid at the bottom, probably due to milk, which is his main support, but of which he has had none since last night. It is one of the most important things to be remembered in examining a patient's vomit, not to take food particles or fluids for abnormal or pathologically significant constituents.

The smell of the material is not unpleasant. Instead of the rancid, sour, fat-acid smell, that sets the teeth on edge when the vomited material has come from the stomach, we have here a not unpleasant coffee odor, and nothing more. There is in it none of the other signs of digestive alteration, no coagulated material, despite the presence of considerable milk, and only mucus, not gastric juice, has been mixed with it evidently, since all the reactions of gastric juice are lacking in it.

The difficulty of swallowing points to a stricture of the cesophagus; the bringing up of material that has been swallowed a good while after swallowing and yet without the changes that would have occurred had it been in the stomach, points to the presence of a diverticulum of the cesophagus. If, now, we listen over the course of the patient's cesophagus, here along the spinal column to the left we find that the ordinary swallowing murmurs are disturbed. I can follow the fluid to a point about corresponding to the bifurcation of the trachea, but there it ceases. This method of auscultation of the cesophagus is often of use to confirm the other signs of stenosis, as it can be done without disturbing the patient and without the slightest danger. You notice how obstinately our patient coughs. There exists almost a constant tendency to cough. Occasionally with the cough he brings up portions of the milk he tried to swallow. It is, as you see, absolutely unchanged. Since taking the liquid the cough is even more frequent than before.

To really judge definitely of the presence and location of a stricture of the œsophagus, we must determine by means of sounds where and how great the obstacle to swallowing is. The one thing to be carefully decided beforehand is that it is not an aneurism which by pressure is causing the narrowing of the œsophagus. The warning is all the more to be heeded as, even in the hands of some of the best clinicians, fatal perforations of aneurismal sacs by a sound have taken place. Here we have absolutely no symptom or physical sign that would point to aneurism.

We pass, then, a medium-sized sound. I prefer for this the flexible English sounds which easily bend on being warmed. I select one of twenty-four millimetres in diameter, and after dipping it in glycerin pass it. In the ordinary passage of a stomach-tube I use only plain water, because of the unpleasant taste of oil or glycerin. Here, however, apprehending difficulty, I anticipate it in the hope to lessen friction. At about twenty-three centimetres from the teeth I come upon an obstacle. This, according to the table of measurements of the œsophagus, is almost exactly at the bifurcation of the trachea, and, comparing the distance by the sound on the outside, which I always consider more satisfactory, since the length of men's necks and, as a consequence, that of the cervical portion of the œsophagus is most variable, we find that the obstruction is slightly above the second costal cartilage. This confirms the measurement as taken from the table of œsophageal distances.

A smaller sound goes somewhat farther, showing that the upper end of the stricture is funnel-shaped, but that, too, is caught and cannot pass. Naturally we have to be extremely careful in our manipulations. We suspect, from the rapid development of the cachexia, the age of the patient, and the absence of other causes, a cancerous stricture. Cancers here are often ulcerated, soft, succulent tumors, and in either case easily perforated. It is very probable that patients have had their lives shortened by careless sounding under such circumstances. In the present case I should not care to pass sounds as a mere demonstration, but our patient has been unable to swallow for some days, and we hope to find and dilate the narrowed lumen, if possible.

Even after a cancerous stricture of the œsophagus has once become impermeable the case is not absolutely hopeless, nor must a gastrostomy be immediately done. Such closures sometimes occur

suddenly. There seems to be a spasmotic contraction added to the neoplastic stricture. Then after a while this lets up and liquids may again be taken. Some such thing seems to have happened in our case. Sometimes the recovery of permeability comes after the coughing up of portions of the tumor; sometimes it seems to follow a gentle dilatation with the sound, which has relaxed the spasm. In some cases no reason can be found for the remission of symptoms that so fortunately occurs.

The passage of the second sound farther than the first by some six centimetres indicates that we have to do with a tumor that involves a considerable portion of the oesophagus, and has an upper ring of tissue that is not as impermeable as the second. We do not succeed in getting beyond the stricture, however, even with a sound of eight millimetres, so I shall reserve further manipulation for the wards, where the surroundings are better adapted for what is, after all, rather delicate work, and the patient suffers less from excitement. That we have a cancerous stricture there is every reason to think. The prognosis is, then, the worst possible.

We have some features in the case that point to a serious complication. There is the almost persistent tendency to cough, a certain amount of dyspnoea, a rise in temperature and pulse, and some lung symptoms. Here on the left at the back there is an area of dulness in which bronchial breathing is plainly heard. We have an infiltration of the lung, but from what cause? We have not to do with an ordinary pneumonia, for we have had none of the usual symptoms. We might think of hypostatic pneumonia, but the patient, though extremely weak, has not been kept so persistently on his back as to cause that condition.

I have in other cases not unlike this sometimes thought of spread of the cancerous process to the lung tissue, and have even dared to diagnose it more than once, but it has never been confirmed at the autopsy. The communication of cancer to the lungs from the oesophagus by contiguity is extremely rare. It is much more probable that we have to do with a perforation of the oesophagus into the trachea. This is by no means of infrequent occurrence in cancers of the oesophagus, and constitutes, in fact, a not unusual termination of such cases.

Here the perforation would seem to have taken place some four or five days ago, but to have been extremely small. From this comes

the persistent tendency to cough, which becomes even more pronounced on his attempting to swallow something. Through this perforation has come the infection of the lungs, and the infiltration which causes the physical signs we have noted in the thorax. Usually death after perforation of the œsophagus comes rapidly, but there are a certain number of cases in which the amount of irritating and infectious material that finds its way into the lungs is but small, and the invasion by micro-organisms gradual. In these the inflammatory reaction leads to the pouring out of an exudate which for a time acts as a protective barrier against the spread of the infection. A localized pneumonia results. Some such process seems to be present here, but it is only a question of time until reaction will no longer equal irritant action, and when this equilibrium is destroyed the invasion will be rapid and abscess or gangrene of the lung result, as is usual in these cases. Therapy can only be symptomatic; even without this lung infection, the tumor is too deep in the thorax and is too far advanced to think of operative interference.

THE TREATMENT OF TUBERCULOSIS.

CLINICAL LECTURE DELIVERED AT THE CHILDREN'S HOSPITAL.

BY PROFESSOR J. GRANCHER, M.D.,

Professor of Clinical Medicine, Paris Faculty, France.

LECTURE VI.

GENTLEMEN,—It does not suffice to know which are the best forms of food for the tubercular patient, and which will supply a maximum of strength for a minimum of digestive effort; you must know how to map out a *régime* for these patients. The art of drawing up a programme of meals of such a nature that they will awaken the patient's appetite and then give him strength is a delicate one; if you acquire it you will succeed, but if you do not, you will fail. You cannot, therefore, pay too great attention to all the trifling details into which I shall now go.

Let us take an every-day case of chronic tuberculosis: both apices affected, one more than the other, and breaking down; evening temperature, 38° to 38.5° C.; poor appetite, fair digestion; marked loss of bodily weight; a slow case, in its third year. Nothing that has been advised so far has checked the disease, although the patient has been directed by a man imbued with modern ideas and who has tried over-feeding, open air day and night, etc. During the last few months the patient has lost ground noticeably.

What is there to be done in such a case? This is how I proceed. I cross-question the patient minutely concerning the real (or supposed) effect of the remedies he has been in the habit of using,—creosote, cod-liver oil, arsenic, antipyretics, etc., and their action on the stomach and intestine; for it is quite exceptional that the benefit derived from them can offset their harmful effect. In almost every case you will find that a remedy which at first seemed of use had soon to be replaced by another, either because it hindered digestion or no longer had any effect. I am so convinced of this fact that in cases

such as I have now supposed I begin by eliminating all remedies of whatever nature, explaining that, whereas in the early stages they may have been of service, and may become so again later on, at the present moment it will be advisable to set them aside. You have to give an explanation of this sort to gain the patient's confidence and not to appear to disapprove of the action of the physician who has preceded you. Patients expect to find in a new physician a new remedy, and will be disappointed if he leaves you without it, and with nothing but general advice concerning food and fresh air. You must make him understand that his safety lies in a *régime*, but in a more sagaciously organized *régime* than he has heretofore followed.

I then ask him to go over step by step the details of each of the three or four meals of which he may partake in the day, and as he does so I make a note of each error of omission or commission. Also I ask how these meals are digested. These slight errors are the reason why his *régime* has failed to have the desired effect; we physicians have not only to lay down a *régime*, but to oversee its carrying into effect.

Thus you will find that our patient in question is badly nourished, although he consumes a large amount of food, maybe too much. He will also, in all probability, digest these meals badly, and through unconsciously living in a state of indigestion he has reached his present condition of bad nutrition and fever. You must therefore get him to understand the mistakes he is making quite unconsciously, and explain to him point by point how to nourish himself and digest what he eats. The difficult point is to decide on the starting-point in his new *régime*,—that is, the minimum quantity that a stomach can accept and digest when it has been overworked for a long time past.

The mistakes made by patients left to their own devices in eating are generally the following:

They drink too much liquid while eating, and thus hinder digestion, especially if, as is frequently the case, their stomachs are dilated. Even when there is no gastric distention, they should limit their drinking so as to increase their digestive power. It is not two or three glasses of liquid that they should take at a meal, but *one*; and even that may be too much if they take a cup of black coffee after the meal. It is better for them to drink too little than too much; a wine-glassful taken in sips is often enough. A curious fact is that this habit of sobriety in liquids can be acquired in a few days, and once

acquired they no longer feel the deprivation in the slightest degree. The adoption of this habit will often improve the patient's digestion wonderfully.

When the decrease in the amount of liquid taken at a meal is accompanied by the suppression of red wine, which so many patients take, mistaking the momentary stimulation it gives for an increase in strength, the improvement in digestion will be greater still.

I also eliminate from the *régime* most forms of dessert,—cakes, fruit, preserves. These desserts, mixtures of fatty and sugary substances that are often not fresh, are very deleterious to delicate stomachs, and hinder the digestion of useful food.

With the desserts I also forbid fatty articles of food, although this is contrary to what is generally taught,—that consumptives should eat a great deal of fat,—cod-liver oil, butter, cheese, fried bacon, etc. If these articles were properly digested I should do as the rest do and advise their use; but they are not. Even milk, at meals or at other times, will often have to have the cream taken off, as it will prove too rich. The only fat that can be freely used without fatigue to digestion appears to be the yolk of the egg; this, with milk and a little sugar, can be given for desserts. I think that the hydrocarbons in cereals, feculents, and Italian pastes, together with the albumen of meat and eggs, are all that is necessary for the diet of a tubercular patient.

My aim and guide in the matter of diet are not to fatten the patient, but to restore to him his ordinary strength and his normal weight, or perhaps a little over. I really do not care to see them go much beyond this. Some of them are carried away by this idea of gaining weight, and starting from sixty kilos, or one hundred and seventeen pounds (their normal being seventy, or one hundred and thirty-six pounds), they not only reach their normal, but go to eighty, or one hundred and fifty-six pounds, and even ninety, or one hundred and seventy-five pounds! This may have serious drawbacks, and in one patient I have in mind the overweight acquired brought on a passive congestive condition of the bronchial tubes and lungs that manifested itself by bursts of fine râles around the tubercular foci, or away from them at the base of the lungs. The deposit of fat around the heart was the cause of dyspnœa, cardiac dilatation, and peripheral vascular disorders.

It will be well, therefore, to see that our tubercular patients do

not increase too much in weight; let them only, by means of a nitrogenous diet, come gradually back to their normal weight, which is of course their weight before they became consumptive. If to this they add two or three kilos, or four to six pounds, there will be no great harm, but you must never lose sight of the fact that what is to be desired is *strength*, not *avoirdupois*; it is the *quality* of the tissue that counts, not the *quantity*.

To return to the fatty articles of food, of which we were speaking, and to take up the type of this class, cod-liver oil, when taken in large doses, may do good; but the lowest dose at which it can really accomplish anything (eight to ten tablespoonfuls a day) is rarely reached, and in addition to this its efficacy is greatest in torpid forms of tuberculosis affecting the glands and bones, and it acts better in children than in adults. Generally cod-liver oil does more harm than good; the dose that is tolerated, one or two tablespoonfuls, is of no account, and, in fact, does harm, because, being badly digested, it upsets the stomach.

In addition to the mistakes that are made at all meals indiscriminately, over-drinking, the use of fermented liquids, and partaking of desserts and fatty foods, there are certain *special* errors peculiar to given meals and due to traditions and customs that are most difficult to contend with.

Thus, the first meal of the day is rarely what it should be for a tubercular patient. It is either too light, not substantial enough,—a cup of tea or coffee merely, because the patient feels no desire to eat in the early morning,—or too heavy and abundant, because the patient, having been urged to eat, does so from the beginning of the day. He may not only eat too much, but injudiciously,—a large bowl of milk, milk and coffee, or chocolate, with bread and butter, seven hundred to eight hundred grammes, or twenty-two to twenty-five ounces, in all. This is too much, especially as the stomach, owing to bad digestions of the day before, may be badly prepared for a full meal. In other cases the error of the first meal will be greater still,—bread or cabbage soup! Wine! While in others still the harm done in the early morning will be increased by a meal taken two hours later, when the digestion of the first meal is still in process of accomplishment. Under such circumstances how will it be possible for the noon meal to be tolerated and properly digested!

For my part I lay great stress on the early morning meal, and ad-

vise it to be substantial in a small volume. My preference is for two eggs, raw or soft-boiled, with a small cup of coffee, milk and coffee, or tea; to this can be added a little cold meat,—ham or chicken. If such a meal is taken at eight A.M., that is enough to carry the patient to lunch at 11.30 or noon. When the patient passes good nights of from seven to eight hours of deep, uninterrupted sleep,—which in my opinion is a good sign of proper digestion,—I advise the first meal to be taken at seven A.M., as above; then at ten o'clock a tablespoonful of beef pulp in a teacup of cold bouillon, and the next meal at 12.30.

The important point is that the tubercular patient should be sufficiently fed, but not overfed, in the forenoon, and should reach the hour of the chief (noon) meal in good condition, hungry and prepared to digest a full substantial meal.

But the patient we are supposed to be treating has a rise of temperature in the afternoon, 38° to 38.5° C., and will not be hungry at noon, and it may be desirable to humor this distaste for food. For these patients, therefore, with fever, I do not give the full noon meal, but limit them to an egg, or some roast meat, or, better still, a tablespoonful of beef pulp in some mashed potato. In such cases it may be advisable to change the usually accepted order of meals (the chief meal at noon) and to feed the patient rather in the evening when the fever goes down.

We have become accustomed to make our chief meal in the middle of the day, and when our patients have no fever at all, or only a slight rise, it is well to stick to our custom, so that the indication for the nature of the mid-day meal will be the patient's afternoon temperature. I have often seen a patient's fever come back to normal by rest in bed and relative diet at noon. When this has been accomplished, we can gradually bring their noon meal back to what it should be. This must, however, be done with the thermometer in hand, as experience seems to indicate that afternoon fever may often be caused, kept up, or increased by defective ideas on diet.

This brings us to a very delicate point in the treatment of tuberculosis by feeding. Shall we feed and overfeed them when their temperature is up, or shall we diet them at such times, and how? It is customary with a tubercular patient with *continued* fever— 37.5° C. in the morning, 38.5° in the afternoon—to decrease the food allowance, and this is probably advisable when we have reason to think

that the fever may be due to laborious and defective digestion. Diet should then be tried for a few days anyhow, but a diet that will allow the stomach to rest without weakening the patient. In such cases I act as follows: Seven A.M., a small cup of milk; at ten, an egg; at one, a tablespoonful of beef pulp in a little bouillon; at four, a cup of milk; at seven, a second tablespoonful of beef pulp; and at ten an egg. No bread or any other article of food.

Except in cases where the disease is very advanced or in following an infectious course, or where the stomach is entirely ruined, the temperature soon decreases and disappears. In some cases, even, the appetite becomes quite sharp as the fever disappears. It is then advisable to go gradually back, thermometer in hand, to the over-feeding, and to reach as quickly as may be prudent the double allowance desirable for a tubercular patient,—the “working” and the “recovery” allowance.

But what should be done if the fever continues in spite of the comparative diet? If we keep the patient at this low notch, he may weaken and prove an easy prey for the bacillus; and if we make the diet stricter still, if the fever does not yield, the danger is greater still, and the patient who is sustained by nothing at all will melt before your very eyes.

I think that in such stubborn febrile cases we should only knock off the food when we are *forced* to by the invincible repugnance of the patient, by a steadily increasing temperature, or by vomiting. It is better, when the test decrease given above fails, to go back to the ordinary diet and play it for all it is worth. No doubt we shall then not always succeed, but we shall do so sometimes, and in some cases overcome both fever and tuberculosis. This is our only chance with a patient who has reached such a predicament.

When, however, our patient's temperature is normal all day, and he feels hungry for his noon-day meal after his one or two morning snatches, we must do our very best with the noon meal. Over and above the usual allowance,—eggs or fish, one or two meat dishes, and one of vegetables,—a tubercular patient should take at each meal a supplement of a tablespoonful of beef pulp.

Two tablespoonfuls of beef pulp and two eggs per diem, over and above the ordinary “working” allowance, constitute what I look on as the “recovery” allowance.

The patient will know that the noon meal has been quickly and

properly digested by feeling ready for something more by four o'clock, and can then take a cup of milk. But if this does not succeed it should be dropped altogether, and the patient should wait until the dinner-hour. The patient must not only be nourished, but must sleep; and sleep is no doubt governed by the digestion of all the meals of the day, and particularly the evening meal.

Dinner should therefore be closely watched. No gamy meat, no sauces, no overeating. No soup, one plate of meat (chicken preferred), one feculent, and a tablespoonful of beef pulp. Copious dinners are a common mistake in Paris and other large cities, but for dyspeptics they are most inadvisable, and will be followed by a bad night's rest, as well as by an inferior state of health the next day; not only does one meal depend on the proper digestion of the preceding one, but a patient's condition on one day depends on his condition the day previous.

The physician must therefore pay particular attention to the first and last meals of the day, generally increasing the former and decreasing the latter. A great point is to know at what moment to yield in the programme of feeding at high pressure, and to fall back to a stricter and more reduced diet when the treatment is not going on satisfactorily, to work gradually forward again to the full *régime*. A few days or weeks apparently lost in this way will be in reality a gain in vigor for the stomach.

Circumstances sometimes render these interruptions obligatory. An haemoptysis, for instance, may make rest and diet a necessity for several days, and in women the appearance of the menses may have the same effect. Or, a consumptive may need a few days of rest per month while getting used to his double allowance of food.

Feeding at high pressure has to be closely watched when a patient leaves a bad climate to go to one that is warm and sunny, when it will often have to be modified and lessened when the change is to be made, for fear of acute dyspepsia with hypertrophy of the liver, fever, etc., which will necessitate a milk diet.

The physicians who practise in sanatoria, where they have to deal with the managers and are not in control, have a difficult part to play. The trouble here is the sameness and ordinary quality of the food, which, although abundant, may not be what is required by fanciful stomachs. Some patients put up with it and get used to it, and I do not wish to speak against sanatoria, which are very useful institutions;

but I must say I prefer treatment at home when the patient is sufficiently submissive and intelligent to help me in the treatment. Many do not come under this category, and for them the sanatorium is a necessity, as they will be *obliged* to obey.

In either case feeding at high pressure, carried on successfully to recovery, acts slowly and indirectly. To the patient's great astonishment, the pulmonary lesions are far from yielding the first, and flesh and weight may have been normal for a long while before the local trouble begins to improve. As a usual thing, the physician will find this therapeutic paradox: the general condition improving while the local condition grows worse, and this for several months! It is only a long time after the strength has returned that the pulmonary lesions first cease to advance, then remain stationary, and finally slowly recede. Nothing could be better fitted to improve the omnipotence of nutrition in the victory of the system over the tubercular bacillus.

SURGICAL TREATMENT OF TUBERCULOUS NEPHRITIS.¹

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF MARBURG.

BY PROFESSOR ERNST KÜSTER,

Director of the Surgical Clinic and Professor of Surgery at the University of
Marburg, Germany.

GENTLEMEN,—Our patient for this morning has a series of symptoms that began some years ago and have continued with certain remissions ever since. He thinks himself that his affection dates from a fall which he had six years ago. He is a ship's officer, and fell from the deck of his vessel into the hold, some twenty feet. He landed on his left side, injuring his hand so that, as you see, he is not able to use it properly even now. He was not unconscious after the accident, and recovered from the shock after a couple of weeks. He thinks he had some discomfort here in the left lumbar region for the next year, and then began to notice that his urine was cloudy. He does not know how long, however, this cloudiness of his urine had existed before he noticed it.

His urinary symptoms grew worse, and four years ago, in the Argentine Confederation, he was told, after an examination of his kidneys and urine, that he had kidney-disease. The attack that brought him to the hospital in Argentine had been ushered in with fever. From his description of the treatment his bladder seems to have been washed out, and after some weeks of rest in bed his symptoms improved and he was able to return home to Germany. Here, consulting a doctor, he was told he had kidney trouble, and he was advised to go to Wildungen (a watering place not far from Marburg, to which patients with urinary troubles are often sent). He grew no better, and at length came to the hospital here in Marburg.

So much as to his present illness. His personal history is not

¹ Reported by James J. Walsh, Ph. D., M.D.

without its interest for us. When eighteen (he is now thirty-eight), he says that an opening was made in his back and pus removed. His description leads us to think there was question of an operation for empyema. When about twenty he had gonorrhœa, and had it again some six years ago, but does not think that any trace of it remained. He is married, and his wife has borne children, but is suffering now from parametritis. He has four children living and two that died when young from causes he does not know. The living children are healthy.

His family history is only of importance inasmuch as his father seems to have died of consumption. His mother died of "dropsy." His brothers and sisters are living and well.

Examination of his urine shows that it is acid, of specific gravity 1015, and that the only abnormal constituent in the copious sediment that settles from it is pus; that is, there are no casts to be found, and none but ordinary pyogenic cocci are demonstrable.

There is question, of course, first, whether this pus comes from the bladder or higher up in the genito-urinary tract, since it is entirely too plentiful to come from the urethra. It has been impossible to get a satisfactory cystoscopic picture of the bladder; no matter how carefully and plentifully the bladder is washed out, cloudy urine collects again so rapidly that the field of vision is obscured. We have not been able to see the entrance of the ureters. This persistent cloudiness of the urine speaks, of course, against the possibility of the urinary condition being due to a bladder affection. Owing to the fact that the patient had a gonorrhœa some five years ago, the question of a gonorrhœal cystitis in the case must be the first one disposed of.

Time was when we believed that because the symptoms of a gonorrhœa were in abeyance the affection itself was at an end. Even when the symptoms that remained were pathognomonic but slight, we did not believe much in the possibility of autoinfection, and consequent exacerbation of the condition. Now, however, we know that so long as the slightest symptom remains there is possibility of reinfecting one's self or infecting others from the limited but still virulent focus that persists. It is even possible that with absolutely no symptoms, or so slight that they entirely escape the notice of the patient, and may escape even his physician's notice unless he is on his guard, a nidus of gonococci remains somewhere in the urethral tract for

years, ready to cause reinfection of healthy parts when for any reason the bacteria take on a rejuvenated virulence, or, what is more probable, when the tissues become less resistant.

We might have in a case like this, then, as the result of a cold or some acute kidney trouble that altered the reaction of the urine and caused the suspension in it of albuminous material, thus making it a more favorable culture-medium, a lighting up of a gonorrhœal cystitis, the micro-organisms having been for years present in small numbers in a chronic inflamed spot in the prostate or posterior urethra.

Gonorrhœal cystitis, however, always changes the reaction of the urine and makes it alkaline. Here we have, as we noted, acid urine. The cystitis that occurs with hypertrophy of the prostate sometimes gives neutral urine, but this will occur only when there is no pus. Where we find an acid urine very cloudy and full of pus, we can be sure that the pus comes at least from as high up as the pelvis of the kidney.

I have already said that the rapid recurrence of the cloudiness of the urine also spoke against the possibility of its being cystitic in origin; pus could not be secreted so rapidly from the bladder walls. The amount shows that we have to do with a not inconsiderable purulent process, which is so situated that the pus becomes intimately mixed with the urine during or shortly after its secretion.

A negative proof that we have not to do with gonorrhœal cystitis is the fact that the most careful search of hundreds of microscopic specimens of the sediment of the urine has failed to reveal a single specimen of the gonococcus. Coccii are to be found which, sometimes evidently caught in the act of reduplication, show the diplococcus form, but diplococci within the cells there are none. In fact, no intracellular micro-organisms of any kind are to be found.

Our repeated microscopic examinations of the sediment have been directed to another end, too. We have been looking carefully for specimens of the epithelial lining cells of the kidney pelvis. We have not been able to find them, however, and this would seem to show that the inflammatory process is situated farther still from the periphery of the urogenital tract,—namely, in the kidney substance. It may be, however, that the chronic inflammatory process (we are assuming from the history of the patient that it has lasted now for some four years at least) has led to the desquamation of the cells

of the renal pelvis some time ago, and that the epithelial lining of this portion of the urinary tract has been replaced by inflammatory exudate and granulation tissue.

The next explanation that would naturally suggest itself as to the etiology of so chronic a process as this would be the tubercle bacillus. The most careful search so far has, however, not enabled us to demonstrate the tubercle bacillus in the urine. While the presence of the bacilli, however, makes the diagnosis positive, their absence does not absolutely exclude a tuberculous process. They are found, of course, whenever the tuberculously affected tissues are in connection with the pelvis of the kidney; but it is probable that only a small proportion of the cases of tuberculosis of the kidney begin here. Most of them begin in embolic patches of tuberculous material carried to the kidney from other parts and imprisoned in their passage through the renal capillaries.

Most tuberculosis of the kidney begins, then, in the cortex, and it is only after the process has advanced so that connection with the pelvis of the kidney is made that tubercle bacilli appear in the urine. For this the tubercle must have ulcerated, so that broken down material in which the bacilli are contained is discharged into the renal pelvis. While, then, in advanced cases tubercle bacilli are always present in the urine, in the incipient cases, just the ones in which by resection of the kidney conservative surgery could be of the greatest service, for extirpation of the organ is a serious mutilating operation, the bacilli may be absent or appear but rarely, and so be easily missed.

Despite the fact that we do not find tubercle bacilli, we cannot help but suspect a tuberculous process here. With a history going back over four years, and a tuberculous father, it is impossible to exclude the thought that this renal affection is tuberculous. It may, of course, be a chronic inflammatory or purulent process from stone in the kidney, or from some infective embolic infarct; but we have no history of renal colic, and no history of a severe infective fever with heart symptoms or the like, to lead us to think of infective infarct.

Whatever the condition may be that is present, it should be removed. Whatever its cause may be, it is progressively destroying all the kidney substance, rendering the bladder and lower urinary tract liable to infection, and always putting the patient in the danger

of a generalization of the process, since an intracorporeal infectious focus always exists.

If, as we suspect, we have to do with renal tuberculosis, how did it originate? Is it primary or secondary? Contrary to what we believed some years ago, we are now inclined to think most cases of renal tuberculosis primary,—i.e., at least, in the sense that it is not secondary to any other organic lesion, though the infective material has come through the blood. Just how infection has come may not be so easy to determine, but this is true in most cases of primary tuberculosis. When in a child a gland or series of glands enlarge on the same side of the head as that on which a tooth has just come, or is coming, or not far from some erosion of the mucous membrane of the mouth, or the tonsils, we know whence the infective material has come.

When post-mortem we find tuberculous processes in the spleen, we know that the infectious material must have come through the blood; when we find tuberculosis of the spleen as seemingly the only macroscopic lesion in the body, then it is hard to think of it as primary, for the avenue of entrance of the bacilli is difficult to imagine, since it seems clear that bacilli cannot penetrate through the normal mucous membrane of the intestine. Though we speak of tubercular lesions of the spleen and kidney where no other lesion is to be found as primary, it is probable that they are usually secondary, the original lesion being minute and hidden away perhaps in some obscure cervical or mediastinal lymphatic gland.

This original lesion need not be recent. Nothing seems clearer than that tubercle bacilli encapsulated in connective tissue may remain for decennia in the body, absolutely without producing any symptoms, and yet retain their virulence. When put in the unfavorable conditions for growth, which ensues when the inflammatory reaction around them has shut them off from the system, they take on the extremely resistant spore form and live on indefinitely. The process of caseation does not seem to interfere with their vitality in the least, and they may even retain their virulence in the midst of calcified material.

The mouth and pharynx, especially the tonsillar tissue, give entrance to the tubercle bacilli in early years, and the cervical glands filter them out and retain them, while the mediastinal glands perform the same function for those that penetrate the mucous membrane of

the respiratory tract. Here they remain encapsulated until some inflammatory process in their neighborhood, or some run-down condition of the system, leads to the vascularization of their capsule, when the bacilli may be taken up and carried to an organ or various organs, setting up miliary tuberculosis, when a primary tuberculosis of that organ or those organs seems to be the pathological lesion of the case.

This term primary tuberculosis is true in a certain sense even then, for the old encapsulated tuberculous focus surrounded by connective tissue is practically outside the body, and reinfection from it is as truly a primary infection of the organism as if the bacilli gained an entrance into the body at some other point.

Very often these new tuberculous infections take place after accidents. The general shaking up of the body, or the call upon tissues during the time when the patient is convalescing from an accident, seems to supply the conditions suitable for the reabsorption of the bacilli into the circulation. The presence at some point of an effusion of blood into the tissues furnishes an excellent culture-medium for the bacillus, which grows best, as you know, on blood-serum. Not infrequently, then, in tubercular arthritis is there a history of preceding traumatism.

In our case, then, where we have the history of a fall on the lumbar region, the resultant traumatic condition of the kidney may have proved the occasion for the tubercle bacillus to find a favorable nidus for growth. That a slight trauma of the kidney, giving rise at the moment to practically no symptoms, and yet causing a hemorrhagic exudate, such as we have spoken of above, is possible, no one will doubt.

The next important question for us is which one of the kidneys is affected, or are both? This question in the face of radical operation, the only rational procedure in the case, is extremely serious. To remove a tubercular kidney in a case where the other is in as bad a condition as the one that has been removed, is to shorten life rather than to prolong it. Tubercular involvement of both kidneys is not so rare but that it must needs be taken into account in every case where there is serious question of unilateral nephrectomy, and no conservative surgeon would neglect so important a precaution.

Then it may happen anomalously that the other kidney is absent or is so small that it cannot be expected to take up the function of

the sister organ, in case it should be removed. An operation with radical extirpation will in such a case be inevitably followed by death.

The condition of the other kidney is not easy to determine. The diseased kidney may give practically no external symptoms. In our case here, deep palpation in the left lumbar region shows that the kidney on the left is tender. Very little can be concluded from this, however, as at times where one kidney is much diseased and the other practically doing the work of both, it is the unaffected one that is tender on palpation; the compensatory hyperæmia and the consequent distention of the capsule of the kidney causing this.

If we could with assurance catheterize the ureters of all our male patients, then it would not be so difficult to decide which kidney was affected, and whether the other was in good condition and secreting a normal amount of urine; but we cannot do it in many cases, and in a case like the present, where there is deep cloudiness of the urine, persistently recurring in spite of repeated vesical lavage, the attempt is practically sure to be hopeless. The cystoscope, by showing the condition of the urine from each kidney as it issues from the corresponding ureter, is ordinarily of the greatest service for the diagnosis of which kidney is affected; or, if both are, which most seriously. In our present case we are deprived of this aid also.

There remains, then, the plan suggested by Rovsing, of Copenhagen,—the exposure of both kidneys before extirpation, in order to decide, first, that there is another kidney; second, that it is of a size to justify the idea that it will compensatorily take up the work of the absent kidney,—*i.e.*, that it is not anomalously small; and, third, that it is not diseased. Lawson Tait suggested this bilateral examination of the kidney before extirpation in the old operation of nephrectomy through the abdominal wall, in which the peritoneal cavity is opened. In this, of course, there was no need for a second special external incision in order to expose the other kidney, but the method has been abandoned for the retroperitoneal route because of the danger of general infection of the peritoneum, at least, if there is question of an operation on a kidney disorganized by an infective process in it.

The question of making a second lumbar incision in order to ascertain the condition of the other kidney is no longer a merely theoretic one, but has been put into practice now a number of times. It is practically without danger under proper aseptic precautions, and

the manipulation to which the healthy kidney is subjected has not proved, so far as practical experience up to this time goes, liable to disturb the function of the organ. As it is extremely important that its functional capacity should not be lowered, since it is to be called upon to replace the other kidney entirely, manipulations should be carefully made, but still should always be sufficient to enable the surgeon to be assured of the condition of the kidney.

As the kidney to be extirpated has usually for some time been either functionless or doing very little of the urinary excretion, the other kidney will be found often compensatorily hypertrophied and more hyperæmic than normal. This must not be taken for a pathological condition, but is really the best assurance that the organ is in a proper state to take up the double excretory function that will be demanded of it.

If we find the other kidney normally healthy then will come the question of extirpation. If we obtain assurance of our diagnosis of renal tuberculosis on inspection, then we shall remove the whole kidney. There can scarcely be question of leaving any part of a tubercular kidney, even though it should seem macroscopically healthy. The danger of recurrence or rather of further spread of the disease from unrecognized infectious foci is too great. If another infective process is at work, however, an abscess limited to a certain portion of the kidney, and we have not been able to absolutely exclude this in our diagnosis, then we must judge of the advisability of resection.

We may remove a triangular portion of kidney substance, securing bleeding points by sutures bringing the tissues together, and getting healing by primary union. If we find calculi in the kidney, despite the absence of history pointing to any symptoms of renal lithiasis, then we may resect the kidney for their removal, or even lay the kidney open somewhat as they do for pathological demonstration, and yet preserve some of the organ and retain its excretory function for further usefulness.

The attempt to remove a portion of the kidney for tuberculosis would be foolhardy, since almost certainly smaller foci of infection in the pelvis and other parts of the organ would be left. Very often in tubercular cases the ureter is also affected in these cases. The first operators who realized this thought it necessary to remove as much as possible of the infected ureters. They were even followed

all the way down to the bladder, so as to be radically extirpated. In an extremely bad case, however, in which my assistant, Professor Basch, seven years ago, found not only the ureter, but the adjacent connective tissue infected and his patient extremely weak, he thought it very unadvisable to attempt so serious an additional operation. He left the infected ureter and packed the wound. The result was a fistula that healed after a time and left the patient absolutely without symptoms. He got married later, had healthy children, and was able to go about his work perfectly well, even down to the present time.

The removal of the principal focus of infection, then, may be sufficient. Nature, especially after the constant irritation of the passage of urine has been done away with, will usually be able to take care of what is left of the tuberculous process by herself. Partly by its separation and discharge through the fistula that remains and partly by its encapsulation, the tubercle bacillus, if present, will be effectually prevented from giving further symptoms or spreading to surrounding tissues.

I do not say this to encourage you to do partial operations, for in tubercular processes resections that leave any portion of infected tissue always carry with them the serious danger of renewal of the activity of the affection after a time. But I wish to impress upon you the fact that with an exhausted patient—and patients who have been suffering for years from tuberculosis of the kidney are often extremely unfavorable subjects for long surgical operations—you must not feel yourself absolutely bound to go on and remove all of the infected tissue, even though it may be tedious and exhausting, as if, otherwise, you would do your patient no good. This is an extreme opinion, and experience has shown that in operations upon the urinary tract the removal of the tubercular tissue readily reached may be followed by complete cure even though some infected tissue remains.

In this case, after careful washing out of the bladder until the contents come away perfectly clear, we shall make first our exploratory incisions. In cases where the process seems to be limited to one kidney, and where we have assurance that that kidney is seriously affected, then I usually begin with exploratory incision over what I hope to find the healthy kidney. The asepsis is surer, and the wound may be at once closed up and screened from danger of infection by

careful bandaging. If this kidney proves to be affected, and the catheterization of the ureters or the cystoscope has shown for sure that the other kidney is affected, then the operation may stop here.

In this case, however, we shall begin with the exploration of the diseased kidney, because, as I have said, we have found no pathognomonic signs of tuberculosis, and so may find a condition of renal lithiasis or localized abscess of the kidney, for which nephrotomy or incision may suffice, and then the exploration of the other kidney will not be necessary.

The exploratory incision on the right showed an enlarged, somewhat hyperæmic kidney, which was evidently already doing a considerable amount of compensatory work for the other kidney. It was replaced and the wound carefully closed and sealed. The other kidney was found very extensively diseased, the pelvis having become a pus-sac, in which were found several large stones, evidently the cause of the whole process. Nephrectomy was done and the wound closed.

[NOTE.—Six weeks after the operation the patient was able to leave the hospital perfectly well. His urinary symptoms had completely disappeared and he was passing a normal amount of clear urine of about normal specific gravity.]

THE DIAGNOSIS, MANAGEMENT, AND TREATMENT OF PLEURISY WITH EFFUSION.

BY LOUIS FAUGÈRES BISHOP, A.M., M.D.,

Chairman Section on Medicine, New York Academy of Medicine; Attending Physician Colored Hospital; Assistant Physician French Hospital, etc., New York.

THERE is no condition more interesting than pleurisy with effusion, nor is there one that is capable, under certain conditions, of presenting greater obscurities of diagnosis or stronger reasons for the active intervention of the physician. The diagnosis of effusions into the pleural sac presents problems in physical exploration that are not always successfully solved by those who have not realized the possibility of error even in cases that are apparently quite simple. In all chest diseases, fluid should be made the subject, at frequent intervals, of diagnosis by exclusion. This is particularly necessary in those pulmonary conditions which are liable to become complicated by effusion either simple or purulent.

Fluid is only found in the pleural cavities as a result of some disorder of the body. It is never found in healthy individuals. The cause of fluid may be local disease of the pleura itself, or it may be the result of the conditions that produce dropsy in other parts of the body. The conspicuous causes of fluid in the pleural cavities are pleurisy and disease of the kidneys.

Cardiac disease does not usually cause effusion until the kidneys have been involved by congestion or inflammation. Fluid is more common in acute inflammation of the pleura than when that inflammation has become chronic. The physiology of the pleura is not different from that of the peritoneum in that it seems better able to take care of itself after repeated attacks of disease than when inflamed for the first time. The only condition included in this discussion is the non-purulent effusion. The importance of a serous effusion into one or both of the pleural cavities depends primarily upon its amount. A small effusion occurring in the course of kidney trouble does not

influence the condition of the patient at all. A large effusion occurring, perhaps, from a true inflammation of the pleura may be apparently well borne, but its mechanical removal is indicated on account of the possibility of sudden death. When the quality of the fluid is altered and the pleural cavity is converted into an abscess cavity, the importance of removal becomes imperative. The diagnosis of fluid in the pleural cavity depends upon its physical characteristics as compared with those of the surrounding tissues. Fluid when enclosed in an elastic capsule forms a body that when percussed offers a certain resistance to the finger, that does not transmit sounds, and does not convey sounds from neighboring bodies.

Sound may be conducted along the chest wall from neighboring parts of the chest, or there may be adhesions producing conducting media, just as if there were a cross-piece in a bag of fluid. Thus it results that, while in a simple effusion that does not last long there may be found the classical signs of fluid in the pleural cavity, yet in many instances the signs are so complicated by the eccentric conduction of sound on account of the great irregularity of the sac containing the fluid, that a final conclusion is only reached by the use of an exploring needle. Strangely enough, though skill in diagnosis actually does improve in those who have particular opportunities and give special attention to physical diagnosis, it is found these same practitioners suspect fluid in many instances where others do not, and insist most emphatically upon the necessity of the exploratory puncture to confirm its existence.

The physical diagnosis of pleuritic effusion must depend upon an accurate knowledge of the physics of the chest and an extended experience of diseased conditions. The classical signs are flatness on percussion and the absence of vocal fremitus and breath sounds. However, these signs may be very different in any but the simplest and most recent effusions. Of all the signs, flatness on percussion is the most constant.

The presence of fluid gives a stony resistance to the percussing finger that at once leads to a careful consideration of the other signs. The possibility of insidious accumulation of fluid in the chest should never be forgotten. When the fluid accumulates gradually there is no lesion in the body so extensive that may give so few symptoms. Many patients, when asked to breathe, go through the motions of respiration without allowing very much air to enter or leave the

chest. In some this is the result of actual feebleness of respiration. Particularly is this true towards the base of the lungs. The diagnosis is often half made when we have succeeded in hearing the breath-sounds at the base.

The physical signs of fluid in the pleural cavity are not, therefore, such as can be categorically stated. It is my experience that bronchial breathing, sometimes of a particular type and sometimes not, is as often found as the absence of breath sounds. The percussion note is sometimes even tympanitic, and, moreover, all these signs may be simulated by a consolidated lung, a thickened pleura, or simply by a lung that is not properly expanded or is slightly œdematos. In the detection of fluid the quality of the physical signs is of great importance. The breathing, when not absent, has a peculiarity of its own, and the vocal fremitus disappears in rather a characteristic manner as the chest is examined from above downward. The habit of making in all instances a complete exploration of the chest, going systematically over healthy and diseased portions alike, gives the faculty of judging the relative importance of signs. When all is said, there will be many cases that must remain obscure, and in these the exploratory puncture should certainly be made.

The advantages of the exploratory puncture are those of a certain diagnosis in any condition. The dangers are from infection, as in a case where I saw at an autopsy a suppurating sinus connecting with an abscess of the lung at the site of an exploratory puncture. Hemorrhage may occur when an artery or other important structure is injured. I have seen at an autopsy distinct evidences that the needle had abraded the surfaces of the heart. This produced no bad symptoms, but the possibilities were not pleasant. However, were the disadvantages greater, the advantages of the exploratory puncture would more than outweigh them. The needle is entered at the most promising point. No anæsthetic is necessary, as the pain is but trifling if the needle be introduced with a single steady thrust.

The treatment of pleurisy with effusion by drugs does not afford a brilliant example of success in therapeutics, though spontaneous recovery is frequent. It has generally been undertaken with a view to increasing the elimination of fluid from the body by diuretics and purgatives, and by counterirritation of the chest.

It is doubtful whether these have much effect upon pleuritic effusions. The reason for the effusion is something more than an

accumulation of fluid in a particular place, because the circulation is overcharged with watery matters. There is a local reason whereby the natural exudation of the pleuritic surface has been increased and the natural absorption diminished until the balance has been in favor of accumulation. The specific gravity of blood-serum is maintained by so delicate a mechanism whereby excretion follows the slightest excess and thirst the slightest need of water, that it has never seemed philosophical to me to expect to affect an effusion depending upon local inflammation by any possible amount of change in the circulating blood. Still there is good authority for a dry diet and restricted fluids. The condition seems to me more like a tank on the bank of a river. The river is ready at any time to receive the water from the tank when it is released, and no amount of clearing of the channel of the river or increasing the volume of its flow will be of any advantage to the emptying of the tank. Or, furthermore, if the tank is to be filled from the river the disproportion in amount of water involved is so great that short of entirely drying up the river the sufficiency of water to fill the tank would not be removed. So it would seem that, beyond the indirect influence of the general hygienic improvement that comes from active elimination, pleuritic effusions are not materially affected thereby. Iron, in improving the quality of the blood, is of undoubted value.

The treatment of pleuritic effusion, aside from mechanical intervention, consists in putting the patient in the best possible condition for the absorption and elimination of the fluid. Every-day experience shows that dropsical effusions are much more rapidly disposed of when the body is at rest and when the diet is a simple one. Thus how often have we seen patients brought to the hospital with swollen feet, more or less anasarca, hyperæmia, and a good deal of moisture in the lungs, who in the course of a few days, by simple rest in bed and a milk diet, recover completely from any signs of exudation. It would seem that when the forces of the body are not exhausted in other directions they take to themselves the task of readjusting the hydrostatic relations of the tissues, so the first thing to insist upon with a patient with pleuritic effusion is absolute physical rest and a plain diet of moderate quantity. The next point of routine practice should be a proper supportive treatment to the action of the heart. The danger of heart failure in pleuritic effusions is not an imaginary bugbear. The cases that I have seen of sudden death with pleuritic

effusions have impressed me with the fact that these deaths were due to some inhibitory force acting upon the heart. The close connection of the pleura with the nervous system is something astonishing in particular instances.

There are a number of cases on record where temporary hemiplegia has followed repeatedly the washing out of an empyemic chest.

Passing from the medical treatment of fluid in the pleural cavities, we cannot do better than discuss the general operations of paracentesis. In these days aspiration has almost completely displaced the simple puncture used by the ancients. The advantages of aspiration over simple drainage are the smallness of the aperture, the almost certain prevention of the entrance of air into the pleural cavity, the power of withdrawing fluid even when under negative pressure, and the ability to empty the pleural cavity even when the puncture is not at the most dependent portion of the sac. The latter point is an interesting one. In simple pleurisy it would be possible, with the instruments that I consider the best, to empty the chest, even should the puncture be made above the level of the fluid. This is easily demonstrated by a simple physical experiment in which a collapsed bladder, representing the lung, is hung in a bottle connected with the outside air. Now, this bottle may be emptied through another tube near its top by aspiration, the bladder expanding to replace the water. These are the same conditions presented in the chest. The fluid may be aspirated from the top of the chest, the lung expanding to take its place. The best aspirator is the Dieulafoy. I prefer it to all the smaller and less powerful instruments, which, as every one knows, are apt to fail at the critical moment.

This instrument was devised at a time when aspiration of the chest excited more interest and attention than it does now. Since that time poorer and feebler instruments have been introduced without causing the protest that they would have raised at a time when aspiration was more under discussion. A small instrument exaggerates the importance of the slightest imperfection. When the vacuum cavity is small a few bubbles of air will make it relatively feeble, while with a large vacuum a little leakage is not important. There is, of necessity, a little air in the tubes and needles. For aspiration of clear fluid a small trochar and canula so constructed that the trochar can be withdrawn without disturbing the connection or allowing the entrance of air, is best. For this purpose the trochar works

through a packed collar, and there is a side tube to the canula for attachment of the aspirator. I have always wondered why it is so difficult to purchase these. I think the reason is that the instrument-makers provide themselves with an assortment of sizes, and the first physician who comes along as a purchaser gets the smallest and best. At any rate, it is necessary to have these small instruments made. The trochar is about the size of a knitting-needle. It has an extra aperture near the end, and is, of course, in perfect condition.

A great advantage of the efficient aspirator is that it can be used with a smaller needle than a feeble one. A good place to aspirate an effusion is about two inches below the angle of the scapular, but the exact spot is always best chosen by a careful auscultation and percussion at the time of the operation. It is a good plan to explore with a very fine needle before entering with a larger instrument.

Aspiration of the chest is not the simple procedure that it seems when performed by skilled hands, and it is only accomplished smoothly and safely with satisfactory instruments and well-worked-out antiseptic details. I have had experience with nearly all kinds of instruments, and the percentage of cases in which the apparatus gave out in some detail at the critical moment was very large. Not only was this true when the aspirator was old, but also in at least one instance when the aspirator that a gentleman had just brought from Paris was new, but had lain for some time unused. Attention should be given to the arrangement of the patient. Anæsthesia is not generally necessary. It is frequently undesirable. The patient is supported in a semi-prone position by means of a bed-rest, which is so placed that half of the chest extends over and can be reached from behind. The aspirator is placed on a small table beside the bed, and is managed by an assistant. Just previous to operation the point of puncture is percussed and auscultated, to be sure that the spot promises fluid. This point is marked with a pencil, and the skin, which had previously been sterilized, is washed again with an antiseptic solution. Now the stethoscope is laid aside, and the hands of the operator are thoroughly cleansed. The trochar and canula are attached to the aspirator. The index-finger of the left hand is used to feel for the intercostal space, and then the trochar introduced with a decisive thrust. It is claimed by some operators that the introduction of the trochar is much facilitated by oiling it, but the well-known tendency of oil to carry infection has made this always seem to me undesirable. As

soon as the trochar is in place the connections with the exhausted cylinder of the aspirator are opened and the trochar is withdrawn. This is the critical moment in the operation. If the fluid flows clearly and evenly, we have only to proceed to withdraw in a deliberate and cautious manner as much as is desirable. The causes that may bring about a failure of obtaining fluid when aspirating would well form a discussion by itself. The canula may have become blocked or the chest wall may not have been penetrated, the costal pleura having been pushed in front of the instrument. There may have been an adhesion just at the point of introduction, and the instrument may have entered the lung.

The danger most feared in aspirating is syncope. For that reason a patient should always be given a stimulant before the operation, and during the operation the pulse should be carefully watched. At the slightest signs of syncope the operation should be suspended and the instrument withdrawn.

We have in the diagnosis and treatment of fluid in the pleural cavity a subject well worthy the attention of all general practitioners. We find that this fluid occurs as the result of either general or local causes, that its importance depends upon its amount, how well the patient bears it, and whether or not it is purulent. We find that its diagnosis is founded upon physical examination, but it is frequently necessary to use the exploratory puncture to confirm its presence. The exploratory puncture is not attended by much danger, and is justifiable in nearly all doubtful cases. The best instrument for removing a large accumulation of fluid is the large-sized aspirator; the best instrument of puncture is the trochar and canula, so arranged that the trochar can be operated without the admission of air. The point of aspiration of the chest is a matter of choice. The precautions and dangers are those that are involved in any surgical operation, and are not to be made light of. Death during this procedure is rare, *but from its neglect, frequent.*

THE TREATMENT OF RECENT AND OLD FRACTURES OF THE PATELLA.¹

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF GREIFSWALD, GERMANY.

BY PROFESSOR H. HELFERICH,

Professor of Surgery and Director of the Surgical Clinic at the University ²

GENTLEMEN,—Fracture of the patella has always attracted a great deal of attention from surgeons, and especially from those who had a particular interest in bone surgery. It has been so because, despite the almost innumerable contributions to its treatment, there are still a certain number of cases where the results obtained by our accepted methods of treatment are anything but satisfactory.

One reason for this failure to secure good results, and the main one, is that fractures of the patella properly belong to the class joint-fractures,—i.e., bone lesions complicated by a simultaneous lesion in a neighboring joint. In even the simplest fracture of the patella there is always some injury of the knee-joint. Usually there is an immediate hemorrhage into the capsule of the joint, and later there is a serous effusion from irritation of the serous membranes of the joint. A simple fracture of a long bone may be treated schematically according to certain definite and well-understood indications, but a joint fracture is always a something apart, requiring special study for itself. For an ordinary fracture you know my rules, which are the universal ones: We (1) set it; (2) retain the fragments in place by splints and bandages; (3) keep it from movement until firm union has taken place. Fractured limbs should not be kept too long at rest, as this often leads to atrophic changes in muscles from disuse, and this atrophy is sometimes long in disappearing, or leaves a

¹ Reported by James J. Walsh, Ph.D., M.D.

² Since the delivery of this lecture, Professor Helferich has accepted the call to the Chair of Surgery at the University of Kiel, made vacant by the resignation of the well-known Professor Esmach.

persistent weakness, or, at least, awkwardness in the use of muscles behind it. I generally leave fractures of the limbs four weeks, not longer.

I do not approve of the practice adopted by some surgeons of leaving fractured limbs for a long time in dressings without looking at them. Those who leave fracture-bandages undisturbed for three, four, or five weeks must sometimes have cause to regret their inattention, and one such case of unsatisfactory union is more than enough to make up for the additional slight loss of time and inconvenience for doctor and patient which the more frequent dressing of fractures and the consequent keeping them under observation involves. On the other hand, I must warn you against that meddlesome interference with fractures which is apt to be the fault of the young surgeon in his first fracture cases. The first dressing, if attended by no inconvenience to the patient, may be left for eight days; the second for eight days more; the third may be left for two weeks, by which time the fracture will usually be sufficiently firmly united to permit of prudent usage of the limb. The second dressing, eight days after the fracture, is often a very important one. It permits of the correction of errors in the reposition of the fragments, which may have taken place because of the swelling of the soft tissues and the inflammatory reaction of the parts shortly after the accident, for these inconveniences will by this time have subsided.

In joint-fractures the procedure is not so simple. First, reposition and retention are not so easy as in ordinary fractures, and secondly, there is danger, in long immobilization of a joint with an inflammatory process near it, that adhesions will be set up and ankylosis result. For fractures near the elbow, for instance, in the hope of avoiding this inconvenience, we dress it first in an obtuse angular position, then at a right angle, then at somewhat of an acute angle. Just as soon as the fragments are united—*i.e.*, in from twelve to fourteen days—we begin passive movements with massage, the medico-mechanical treatment as you are familiar with it in our wards. This gives much better results than the old method of keeping the parts absolutely at rest, which was so often followed by an immovable joint.

In fact, it is the better results secured by the immediate commencement of passive movements in such cases, as compared with the old complete immobilization, that have been the main support

for the treatment of fractures by the immediate institution—*i.e.*, within a day or two after the accident—of massage and gentle movements, a method that has attracted a good deal of attention of late. Here, however, as in most things in medicine and surgery, “*in medio tutissimus ibis.*” The safest practice is the middle course, the immobilization of fractures of the shafts of bones, massage, and passive movements to prevent pathological adhesions in joint-fractures, only after physiological union of the fragments has taken place.

Besides the joint complications, fractures of the patella have this peculiarity, that while the displacement in ordinary fractures consists in an over-riding of the fragments, in patellar fractures the dislocation is *ad longitudinem*,—*i.e.*, the fragments are separated from one another. The upper fragment is pulled upward by the quadriceps femoris attached to it. The main indication for treatment is the bringing of the fragments together, which, as a rule, is done without difficulty, but their retention in exact apposition makes one of the most difficult questions in the treatment of fractures. When fracture of the patella occurs without rupture of the tendon, in which the patella is situated, it heals without any difficulty or complication.

It is to be remembered that the patella is a sesamoid bone developed in the tendon of the quadriceps, and that a fracture of the bone without complete solution of continuity of the tendon would, especially where subjects are not beyond middle age, be theoretically possible. That such fractures can and must occur in practice is a well-recognized fact, and you can see the picture from the dissection of such a case in my “Atlas of Fractures and Dislocations” (Lehmann’s series of atlases), which I pass around. Such fractures need, however, no special care beyond rest, and are often unsuspected, the injury being considered to have affected rather some of the tissues of the joint itself. Of this variety of fracture, then, we shall not speak further than to say that they are usually caused by direct violence, may be detected by crepitus, and should be carefully differentiated from so-called sprains of the joint.

In fractures of the patella with longitudinal separation of the fragments three factors tend to make the persistent apposition of the fragments after reposition is secured often very difficult.

1. First, when the aponeurosis of the patellar tendon is completely ruptured, the powerful muscle, the quadriceps femoris that it is inserted into, meets with no opposition to its contraction, and as it

has usually been irritated by the injury to the leg, a state of spasm of the muscle ensues which is extremely hard to overcome and effectually keeps the fragments apart.

2. The second element that renders apposition of the fragments difficult is the forcing up of the capsule of the joint and the overlying soft tissues between them, by the hemorrhage which takes place into the joint so often in such injuries. This is complicated later by the occurrence of serous effusion into the joint, which tends also to force the soft tissues into the artificial space between the fragments of the patella, where a state of lowered pressure exists, and where the capsule of the joint meets with the least resistance to its expansion under the increased fluid within it.

3. The third annoying factor in these fractures has been pointed out by the distinguished Scotch surgeon, MacEwen. He demonstrated by dissections that in fractures of the patella a fringe of aponeurotic tissue from above the fracture might easily be carried between the fragments and retained there, thus preventing apposition. Immediately above the patella, that is, more superficially, there is a layer of cartilage that, being brittle, always breaks with the patella. Above that there is a layer of aponeurosis, which is fibrous in character and somewhat elastic. This does not always break with the patella itself, but is gradually torn across afterwards from over-stretching of its fibres in efforts at extension of the limb or when it has been for some time on the stretch. This gradual process of solution of continuity leads to the formation of a fringe of tissue, not unlike the fringe of hair young girls wear and which is supposed to make them pretty. This aponeurotic fringe serves no such æsthetic purpose, but, dropping between the fragments of the patella, prevents their exact apposition and interferes with union.

There are certain other factors which have been mentioned as delaying union of the patella,—*e.g.*, the normally poor blood-supply to this sesamoid bone, the fact that there is only a thin layer of periosteum here to take on itself the duty of forming the complementary new bone, etc., but these do not explain why, while some fractures of the patella do not heal satisfactorily, others do with excellent results.

The important disturbing factors whose elimination must be secured are the three mentioned,—1, the spasmotic contraction of the quadriceps and separation of the fragments; 2, the interposition of

soft tissues, first, by internal pressure from bleeding into the joint, and, second, from the fringe-like rupture of the aponeurosis of the quadriceps tendon.

The presence of these disturbing factors is, as can be readily imagined, not always easy to detect. Even after detection their removal is not easy. Some surgeons have gone so far, then, as to suggest, in practically all cases, the cutting down on the patella, the careful removal of all soft tissues from between the fragments, and their retention in accurate apposition by some mechanical means, suture or wire. As many fractures of the patella unite very well without any such radical means, and as the proximity of the joint introduces an element of serious danger in case of the slightest fault of aseptic technique during the operation, this seems hardly justified for all cases. On the other hand, where patients are young, or where, owing to their occupation, the function of their patella is important to them, then I believe, if we have any reason to suspect the existence of any of these complications, or if bony union is delayed, that we should cut down and mechanically unite the fragments.

In general, however, it seems best to treat the fracture primarily without operative interference; if, later, function is seriously disturbed by the resultant union, then we may suture the fragments in a secondary operation. In judging of the necessity for this secondary operation, appearances must not be allowed to influence our decision. A most unpromising-looking patella may functionate excellently, and a separation of the patellar fragments by over an inch may still allow excellent use of the leg and perfect function of the quadriceps. The muscle seems by a permanent shortening to have somehow compensated for the greater functional contraction required.

If the hemorrhagic effusion into the joint especially seems liable to keep the fragments of the patella apart, or to disturb the apposition of their surfaces by its pressure, or to push soft tissues between them, then I think there should be no hesitation in puncturing the joint and allowing the exit of the excess of fluid within the capsule.

A prominent factor to be dealt with in all fractures, the spasm of the muscles near the fracture, comes into play here especially. The tendency of the quadriceps femoris to contract, since there now exists no point of support for its extension, cannot be entirely eliminated, but it may be greatly diminished by bending the hip, hence the necessity for dressing the limb so as to lie in an elevated plane. The leg

itself is kept extended, as in this position there is less liability of leverage acting to separate the fragments of the patella, and, besides, in a nearly extended position of the leg there is more room for the accommodation of fluid within the capsule of the joint than if it were flexed, and so the element of pressure from within is as far as possible eliminated.

Despite these precautions and the use of a figure-of-eight bandage, seemingly simple cases of fracture of the patella in which no complication is suspected heal unsatisfactorily. Bony union does not take place, but fibrous, and the fibrous band stretches and injures the function of the limb. The form of the patella after reposition of the fragments cannot always be a guide to us as to the results we may expect, because patellæ have the most varied forms in normal individuals and yet perform excellently their functions. In fact, the form of the sesamoid bone that is developed within the tendon of the quadriceps, in order to carry its action over the angle of the knee, seems to depend to a great extent on the shape of the bony parts and the character of the exercise given it during the developmental period. I have often had the most unpromising-looking patellæ perform their function excellently.

On the other hand, what looks like a perfect result as regards the patella itself may be very disappointing as to function. One great cause for this is that the absolute immobilization of the quadriceps femoris during a long time leads to an atrophy of the muscular elements that is never entirely to be overcome afterwards. For satisfactory function after union it is to be remembered that the conservation of the quadriceps is more important than the obtaining of a perfectly united patella. As the result of this, certain French, English, and American surgeons have adopted the method of beginning almost immediately massage and passive movements for the exercise of the quadriceps in fractures of the patella. But this is to go too far to the other extreme. Immediate mechanotherapy will almost inevitably disturb the proper union of patellar fragments. There is the happy mean here, too. The quadriceps is not to be left absolutely functionless for weeks, but after from three to five days massage is to be instituted, gentle muscle-tapping and stroking downward towards patella, after that mild electrical excitation, and after two weeks careful passive movements without large excursions at the joint are to be prescribed. My personal experience has shown me

that in this middle course, the best results are secured in fracture of the patella. It must not be thought that even recent simple fractures of the patella constitute a type of injury that can always be treated in a certain schematic way. Here almost more than anywhere else in the domain of fractures must the surgeon's judgment and his experience form the guides by which the method of treatment for each fracture is selected, for each of them will have in it something special and personal.

So much for recent fractures of the patella, one of which is to be dressed immediately after the clinic, so that you will have the chance to see the practical application of the principles we have laid down. Now comes the more difficult task of treating old fractures of the patella, that have united with serious loss of function and with considerable displacement of the fragments.

I have two good examples of such cases here to show you. In the one, a man of fifty, there is, from a break of the patella years ago, a space of four inches here between the fragments. Atmospheric pressure forces the soft tissues into this space, and the outlines of the condyles of the femur can be plainly seen through the skin anteriorly. When he walks he really does not use his quadriceps to lift his leg up for the swing forward, though to an unobservant eye he might seem to do so. The limb is pushed forward and partly swung into position by a movement of the trunk and the pendulum effect of the leg. You can notice here in his thighs the difference in circumference between the right and the left, and can see that this difference is mainly due to atrophy of the quadriceps here in front. In a case like this, with the patient of this age and the lesion so old, we could scarcely think of a serious surgical operation. He has grown used to the deformity and the slight halt in his gait that it occasions; he has learned to accommodate himself to it at his work, so that the indications for an operation are scarcely sufficient to justify it. Besides, with the condition as old as this, it is extremely improbable that we would be able to restore the function of the quadriceps, so that the bringing together of the fragments of the patella would have rather a cosmetic than a practical effect.

Our next case is a young man of thirty, in whom fracture of the patella has been followed by fibrous union by a band between the fragments. This band is some two inches long. The function of his quadriceps, too, is greatly interfered with by the amount of con-

traction required to offset this elongation of the distance to the point of application of the force. He is scarcely able to lift his leg when he stands straight, and has very little power in the movement. Here is a case that is eminently suited for the suturing together of the parts of the patella, and it is for that purpose that he is in the hospital, so that you shall have the opportunity of seeing the operation.

As to the difficulties we may meet with in the operation I have a word to say. We may find it extremely hard, even impossible, to bring the upper fragment of the patella down to join the lower one, despite the most careful section of all adhesions. If the patella is already adherent to the femur above, the attempt to bring it down will always be a failure, so that the operation should only be attempted where the upper fragment is freely movable. I must warn you, too, that the forcible bringing of the fragments together and then suturing them, a certain amount of strain being put on the sutures, will always result in failure. The patella is only a sesamoid bone, and never really attains the solidity of ordinary bone, but always retains something of its cartilaginous character. Its consistency is not dense enough to enable it to stand strain on the sutures, and they inevitably pull through. This friability of the patella is still further increased by the malnutrition that follows fracture, so that in old cases it is often very marked and very troublesome to the surgeon.

A division of the tendon of the quadriceps above the upper fragments, though sometimes suggested for these cases, is absolutely impracticable; the discontinuity of the attachment of the quadriceps, from the point of application of its force, is only placed somewhat higher up than before, and no real good is accomplished.

Professor von Bergman's attempt some years ago to bring the lower fragment up, since he could not bring the upper fragment down, was an extremely ingenious attempt at the solution of this interesting problem. He chiselled off the tubercle of the tibia and, having freshened a spot higher up on the bone, made an osteoplastic transplantation of it to this spot. This enabled him in some cases to bring the fragments of the patella together. I well remember, however, at the meeting of the German Congress of Surgeons at which he presented his cases, Volkmann's lively criticism of the method. The keen-eyed experienced surgeon detected at once the fault of the method. The new insertion of the tubercle of the tibia would inevi-

tably interfere with the function of the knee-joint wherever it was really moved up enough to make it worth while to do the operation. Only a few operations of this type have been done as a consequence. A final expedient for the approximation of the fragments might be that a portion of the shaft of the femur be resected in order to facilitate their being brought into apposition. This seems a very serious procedure, but might perhaps be justified. In the leg we remove a portion of the fibula in order to bring the ends of the tibia together when from ununited fracture or necrosis a resection of the tibia is necessary. In the arm, when the *radialis* (the *musculospiral*) nerve has been severed, and the ends have become so separated by contraction that they cannot be brought together, we resect a portion of the humerus in order to facilitate the suture of the nerve-ends and restore the important function of this nerve.

Where but a small amount more was required to bring the patellar fragments together, less than an inch, for example, a resection of the femur well up in the shaft of the bone, at a safe distance from the joint, might be justifiable. The slight shortening that would result in the limb would be more than compensated for by the renewal of the function of so important a muscle as the *quadriceps*. It is to be remembered, too, that exact measurements have shown that the two lower extremities are never of exactly equal length, differences of an inch or more being not uncommon (Treves). We might find in a particular case that this difference was in favor of the leg on which the patella was to be repaired, in which case the resection would be undertaken with even more confidence than usual. This is but another indication of how individual all these cases are and how it is *the particular patient*, not *a particular lesion*, that is to be treated.

Fortunately, however, we do not have to adopt any such radical procedure unless in very exceptional cases. About two months ago I operated before the clinic on a case of fractured patella in which, despite the cutting and tearing of adhesions, and the free use of the periosteal elevator, I was unable to bring the upper fragment down to the lower one. As I had done in two other cases before, I freshened the edges of the fragments and inserted between them several pieces of spongy bone material that had been boiled and preserved in a one-per-cent. solution of sublimate in alcohol. The result you may see in this patient here. He has not a shapely patella, but it

fulfils its function very well. He is not allowed as yet to use his limb with absolute freedom, because the leverage on so large a patella as this is very great, and we wish to be sure that it has become perfectly consolidated before taking any risks of refracture. You can see how much better he can use his leg than this other patient, whom I showed you a moment ago, between the fragments of whose patella there is a space of two inches. This patient is unable to lift his leg, he finds considerable difficulty in going up stairs, and would be absolutely unable to ascend a ladder. Our second patient has an active command of his leg, can ascend stairs, and what is important for him, since he is a painter, will be able to go up a ladder also.

I have had but three cases of this operation as yet, so that an absolutely definite conclusion as to its usefulness and availability cannot be given. All three have, however, been extremely satisfactory results, so that I think we have in this insertion of bone between the fragments of the patella the solution of the difficult question of restoring the function of the quadriceps in these old cases where fibrous union results in such a limited use of the limb.

THE TREATMENT OF TABES DORSALIS.¹

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF BERLIN, GERMANY.

BY PROFESSOR A. GOLDSCHEIDER,

Medical Director of Moabit Hospital and Professor of Special Pathology and Therapy at the University.

GENTLEMEN,—In the last few years the treatment of tabes dorsalis has made distinct advances. Instead of standing nearly hopeless before it while it runs its almost inevitable course, making the patient more and more helpless, more dependent on others, and finally confines him to bed, we are now in a position to so modify its symptoms or restore lost co-ordination that patients are able to help themselves, and devote themselves, even though their tabes itself remains unaltered, to some useful occupation. This is already a great deal gained, and the therapeutics of nervous diseases in the last few years have lost a good deal of their utter hopelessness as the result of the introduction of the compensatory movements.

Meantime, as to the condition itself, the degeneration of the posterior columns of the cord, and its gradual advance, while we have learned that we can, as a rule, do very little, we have found out, too, that there are certain remedies and methods of treatment that have in many cases a definite value.

In a certain number of reported cases of tabes antisyphilitic medication has been of great service. These have been, however, comparatively very rare. As a rule, so-called tabetic cases in which benefit was reported from a course of mercury would seem to have been cases of pseudotabes syphilitica, a luetic affection of the cord, either gummatous in character or due to syphilitic endarteritis with consequent interference with the blood-supply to the cord. Such conditions often give ataxic symptoms very similar to those of true tabes.

¹ Reported by James J. Walsh, Ph.D., M.D.

Mercury, as a rule, if continued for any length of time, would do harm. Chronic intoxication with mercury will of itself produce sclerotic degenerative changes in the nervous system, and will surely heighten any tendency in that direction that is already existent. When a tabes case first comes under treatment it is prudent to try the effect of a mercurial course, for some of the tabetic changes may be due to frank luetic arterial changes, which are capable of resorptive involution. If improvement is not soon noticed, or if the general health or any of the tabetic symptoms get worse under it, it should be forthwith stopped. The same thing does not hold for the iodides, and I am firmly convinced myself that an occasional course of potassium iodide inhibits the sclerotic degenerative changes and hinders the progress of the disease. I have all my tabetic patients take potassium iodide two or three times a year. I begin with from fifteen to twenty grains three times a day, gradually increasing the dose to sixty grains or to the limit of tolerance. I do not believe at all, however, in the large doses given by some, as they are but excreted and serve only to disturb appetite and digestion. A course of the iodides lasts four or five weeks, and then is followed by from six to eight weeks without the drug.

With strychnine I have had some good results. It seems to tone nerve-cells that are lowered in tone, and so keep the degenerative process from advancing. As an experimental demonstration of its action on the vital processes of nervous cellular activity, Flatau and I have shown recently that with the Nissl stain distinct changes in the Nissl bodies of nerve-cells could be observed within two minutes after the injection of strychnine into the neighborhood of nerves leading to them. It was but the scientific proof of my clinical experience that strychnine is capable of wonderful revivifying effects in its action on nerve-cells.

I give .0025 grammie, or about one-twenty-fifth of a grain of strychnini nitrici once a day for three days, and then omit for a day. The injections are continued after this fashion for three or four weeks, and then a longer pause made. I often alternate the courses of potassium iodide and of strychnine, and find, besides the undoubted inhibitory effect of these drugs on the sclerotic process in the cord and on the degenerative nerve changes generally, that the patients, while thus under active treatment, do not so easily give way to the attacks of depression that so often accompany a chronic

nervous disease when the patients become convinced that medication is hopeless. After all, it is our patient we must treat, quite as well as his particular malady, and the saving of these melancholic thoughts is of itself not a little alleviation in the course of the disease.

In certain irritable conditions of the nerve-cells in tabes strychnine is not well borne. If the small doses suggested (and they are purposely small because it is the tonic and not the irritant action of the drug we want) cause slight cramps, or persistent unpleasant muscular twitchings, or tendency to vertigo for some hours after the injections, they should be made even smaller or less frequent, and every third day instead of every fourth may be dropped. If these unpleasant symptoms last only for ten or fifteen minutes after the injection, they may be disregarded. There are great idiosyncrasies to the action of strychnine, however, and these should be borne in mind. Where an increase of pain is noticed, or more frequent or more severe lightning pains, or intestinal or bladder crises, then the strychnine should not be given. Increase of pain is its capital contraindication.

With regard to baths and massage, a distinction must be made as to the class of tabetics for which they are suitable. Sometimes tabetic patients have become obese as a consequence of insufficient exercise due to their ataxia. Such patients are especially suitable for baths and massage. Neither of these valuable therapeutic measures should be employed too vigorously or in a manner to exhaust the patients; gentle massage of the muscles with careful avoidance of all tender points being the indication. In general, obese patients should be brought down in weight, as their general condition will be improved by a quickening of their sluggish circulation, and the movement cure we have to speak of later will be better carried out without the added difficulty of a burden of flesh. This reduction in weight should not be too vigorously pushed, however, as it is exhausting. It will usually be found, too, that the movements we shall describe later had better not be tried during the time of active reduction in weight, but will have a place afterwards.

Often in tabes cases you will find a distinct element of neurasthenia superadded to the tabes. In such cases massage and baths are of the greatest service. It is in these cases of tabes alone, where the neurasthenic symptoms are more prominent than the tabetic symptoms, that you can venture to advise cold baths. Tabetics stand cold

baths very badly, and the baths easily cause more pronounced symptoms to develop, if they do not actually make the pathological condition worse. It is to be remembered that the most prominent thing in the etiology of tabes is exposure to cold, and the sensitiveness to it and to its evil effects seems to remain.

Some tabetics are run down, thin, almost emaciated, one would say, and in such cases, of course, massage and hydrotherapy are out of the question. *The general condition must be improved before anything else is done.* Even attempts at exercises and the movement cure had better not be begun until there has been a decided reaction for the better in the patient's nutrition. In these cases the rest cure with forced feeding (*Mast Kur*) are especially indicated.

In such cases, even when there is no history of a former syphilitic treatment in the case, one should be very careful in using mercury, as the patients do not stand it well. If a mercurial course is tried, as it generally ought to be, for when it does good its success is very marked, the patient should be kept under the best possible hygienic conditions, with nutritious, unirritating, yet plenteous diet, of which milk and the starches and fats should be the principal constituents. They should rest a good deal, have warm baths, should not be allowed much exertion of any kind, mental or physical, and no movement therapy should be instituted till later.

In these thin patients, too, the iodides should be used circumspectly. While I give from forty to fifty grammes of potassium iodide (six hundred to seven hundred and fifty grains) in two or three periods every year to tabetics, and find that it lessens their pains and acts favorably on the arteriosclerosis which so often accompanies tabes, I do not give it to run-down patients. Its depressive effect on the appetite and on digestion more than counteracts in these cases the good it does in other ways.

As to sea-bathing for tabetics, it is, as a rule, not to be prescribed, except with special restrictions and with very well tempered water and climates. As to a sea voyage, sometimes recommended by well-known neurologists, I find the danger of exposure to cold too great, and this, for reasons stated, is, I believe, above all, to be avoided by tabetics.

As to watering-places, I have often found that the baths at Oenhausen and Nauheim do good. In general, however, I think that tabetics are too much discouraged, if they come much in contact with

others suffering from the disease or from other chronic nervous diseases and in a worse state than themselves. Where there is the oft-recurring neurasthenic element in the case this is especially to be avoided.

The artificially prepared carbonic acid baths often do good too. The patients should take them in perfect quiet, as movement drives off the bubbles of carbonic acid gas, while it is its contact with the skin which gives the gentle peripheral nerve irritation that reacts so soothingly often on the central nervous system, and yet is also a gentle stimulant. They are best taken before going to bed, or else should be followed by two or three hours' rest; otherwise unpleasant feelings, at times almost painful in character, develop.

From other remedies that have been sometimes recommended I have seen no good results. Silver nitrate and ergotin do no good. Brown-Séquard's extract of testicle has been given up even in France. Any effects I saw from spermin, its supposed active principle, were certainly due to suggestion, and in one case I found that injections of water, after spermin had been given for some time, produced the same good results as had been claimed for the drug. The phosphates and phosphoric acid have only the same suggestive effects, unless occasionally neurasthenic patients are benefited. When told that they are taking a nerve tonic it invariably does them good.

Suspension I tried a good deal at the Charité, but always was a little anxious as to its possible effect. In heart and arteriosclerotic cases it must not be employed, and it certainly never accomplished the good results for us that are claimed for it elsewhere. I have found Bonuzzi's method, the stretching of the cord and nerve-trunks, that may be accomplished by bending the patient's legs upon his body while in an extended position, often does good for the lightning pains and for the paræsthesia of the legs so frequent in tabes. It does no good for the ataxia, however.

As to the treatment of individual symptoms in tabes, more can be done than for the disease itself. For the characteristic pains, before drugs are used, local measures must be tried. I have often found that heat did good,—*e.g.*, very hot, moist bandages changed every five minutes, or ironing the parts that are painful with an ironer made of metal somewhat like a flatiron and filled with sodium acetate. This is heated until the salt melts, when it retains the heat for a long time, and covered with a woollen cloth may be readily

used for the application of heat. A hot bath, 30° Réaumur (100° F.), with gentle rubbing, which I have called thermomassage, will often relieve the pains.

Heat does good for gastric and intestinal crises too, especially the hot bath. Prophylaxis will often be of benefit here. For the gastro-intestinal crises often following indiscretions in diet relief may sometimes be afforded by a brisk purgative, as Apenta, or one of the other Hungarian bitter waters.

In general, prophylaxis will decrease the number of crises and attacks of lightning pains, as tabetics have such attacks when they overexert themselves mentally, physically, or sexually. You will find that sexual intercourse, for example, will in some patients always be followed by pains or a crisis, and such patients must be warned against indulgence, although all tabetics are not to be absolutely forbidden sexual intercourse. There are some patients who notice no evil effects or depression or exhaustion after it, and in such to forbid would be only to invite infraction of the prohibition and impose needless self-denial.

Where heat does not lessen the pains sufficiently to make them bearable, and unfortunately this happens only too often, drugs must be used, the simpler antipyretic analgesics first, as salicylates, salophen, antipyrin, lactophenin, and various combinations of them; for it is worthy of note that where neither of two drugs relieves the pain alone, two given together may. There is the crossed action of drugs in such cases always to be thought of.

These measures will often not suffice, and then morphine must be used. Subcutaneously is the best, but never allow your patient to have a syringe for himself. Extract of opium may at times replace morphine with advantage. It is more inconvenient for you to have to go each time to give a morphine injection, but it is absolutely the only way to keep him from acquiring the morphine habit. Even with this you will have to be determined and keep your patient thoroughly under your control, or he will acquire the habit in spite of every effort.

For the trophic disturbances of tabes prophylaxis is our best treatment. Tabetics should wear well-fitting shoes specially made for them, not too large, of course, but that cause absolutely no irritation. The first sign of irritation should be the signal for a change, for trophic ulcers on the feet are extremely destructive and often

very hard to heal. Tabetics should be warned, too, at the very beginning of their affection, of the tendency which sometimes develops to easy bone fracture. Here is a man who fell yesterday on his knee and fractured his knee-cap, though it was a simple and not very heavy fall. Unfortunately, this tendency to fracture is itself sometimes one of the first symptoms, so that we cannot give timely warning.

For perforating ulcers and other such trophic changes we find that the playing of a fine stream of hot water on them with considerable force and as hot as the patients can stand it will often cause a hyperæmic reaction that leads to healing. We have had some excellent results from this method, in some cases that had obstinately resisted treatment for years, and we have never seen any bad results. We use the hot spray once a day, and dress with simple sterilized cotton afterwards. This method is also of use in trophic ulcers due to syringomyelia, hysteria, or other nervous disturbances.

And now we come to the treatment of the most important symptom of tabes,—the incoördination,—the one for which I am glad to say we have learned of recent years that we can do most. Incoördination is due to the fact that the muscular sense and the sense of position of the limbs are interfered with as well as other sensory feelings. The result of this is that when movements are attempted they are overdone, and they are not done gradually and continuously, but by little pushes, one after the other. The faint inklings of position sense that come filtering up through the degenerated posterior columns and posterior roots are not enough to enable the will to control continuous movement, but a certain definite distance must be accomplished before it realizes that movement has taken place. The exaggeration of movement is due to the same cause, and the movement must be overdone before the sense tells of its accomplishment.

Now, it is not so clear just how it is done, whether new paths are trained to carry up the sensations, or whether the higher perceptive power is taught to grasp and appreciate by an effort of attention the diminished impulses that come, but it is sure that we can, after exercise, reteach a good deal of the muscular sense and the sense of position that was lost. That is the principle on which the movement therapy is founded,—the regaining of the sense of the exact position occupied by members, joints, muscles, etc. It is not exercise, in the ordinary sense of the word; *that* tabetics, as a rule, do not need, at

least not with the idea of strengthening their muscles, since they are not weak, but it is the accomplishment of *exact* movements that they must be taught.

In general we must first awaken concentration of the attention on the movements. For instance, with this patient here: I have him close his eyes, and then I put his foot in various positions, bending it at the knee and hip, and after each change of position I ask him how it lies. He makes mistakes in telling me, but after a while, by enforced attention, he will be able to translate the faint *inklings* of position sense that come to him better than he does now. He has not the complete sensations he had before his tabes began, but he can learn to appreciate better the significance of the incomplete sensations he still has.

Of course, while moving his limbs I am careful not to let them touch the bed, as his cutaneous sensibility would help him to tell the position of his limbs. But his cutaneous sensibility is also disturbed, as it often is in tabes cases, and exercise will do it good also and generally increase nervous function and conduction. For this I take an electric brush and with a current just strong enough to be felt I touch various groups of nerve-endings, now one, now the other, never staying long in one region, just touching him, not stroking, as that would fatigue and not exercise, and going over the whole body in little jumps more or less symmetrically. This gentle electrical excitation arouses nerve-function. In general, strong currents inhibit, and so we use them for the pains, while the milder currents arouse, as we are doing here. It must always be borne in mind that the degenerate nerve fibres of tabetics easily become fatigued, and that every over-exertion but hastens the degenerative process, so that strong electric currents must, as a rule, be avoided, or the purpose of training the sensory nerve endings at the periphery will not be successfully accomplished.

When we come to the special treatment of incoördination we find two classes of patients to deal with,—those whose ataxia is so far advanced that they are bedridden and those who are still able to be about on their feet. Let us take this latter class first, as it is the most frequent, and as young men particularly you will be called on oftenest to treat it. Here is a tabetic of five years' standing whose incoördination is pronounced. He has the Romberg symptom. He cannot walk along this line on the floor; he widens his base of support by

keeping his legs apart and lifts his feet too high when he walks, turning out his toes.

We shall begin with lessons in stepping and in the placing of his feet. I have him stand and take a step forward, indicating where his foot shall be set down, telling him to do this two or three times, until you see that with repetition he does it better. Now I have him take a couple of steps with the injunction to lift his foot gradually from the ground, the heel first, then the toes, the affected walk of the extreme fop or dandy. This is hard for him, as for tabetics generally, so I place him on this chair and have him lift his heels from the ground several times to get used to the movement. You see how his toes lift almost involuntarily from the ground, and what an effort it is to lift his heels. Now I have him stand up and rise on his toes several times.

You see how he does it: there are a series of little pushes, one after the other, until finally he gets up on his toes. Now this series of interrupted movements, so characteristic of tabes, we must teach him to avoid as much as possible and have him make the movement a continuous one, so we have him repeat this exercise several times.

We have been exercising this patient for some five minutes now, and that is enough for a *séance*. *The concentration of attention* necessary for the tabetic to perform exact movements is very exhausting. Tabes patients often have a lowered sense of fatigue, which makes it easy for them to overdo things, so that we must be ever careful. This diminution of the sense of fatigue is not, however, so frequent as Fränkel believed. There is a sense of intense mental tiredness that develops because of the strain of the attention, however, which is very discouraging and discomforting to the patients and which must be avoided.

After these simpler movements have been practised for some time, and exactitude in them acquired gradually, we go to more complicated ones. Patients practise walking a crack at first with eyes fixed on feet and floor, then with eyes raised, and finally with eyes closed. Then they walk a straight line, placing the heel of one foot immediately in front of the toe of the other. Finally they learn to follow a spiral chalked on the floor, which is of course extremely difficult for them. During these movements they must have either an attendant whose hold gives them confidence, or whose presence reassures them against falls, or they must have a chair or a row of

chairs the backs of which they can grasp if they are in danger of falling. For the spiral and certain other movements a rope swung from the ceiling is good.

They must be carefully guarded against falls, for two reasons,—first, in the lowered trophic condition of the bones a fracture may easily result from a fall, a fall to the knees even easily causing in these cases a fracture of the patella, such as I showed you a few minutes ago; second, the fear of further falls so utterly distracts the attention when concentration of attention is demanded for the exact fulfilment of movements, that very little will be accomplished by the exercises afterwards.

A final movement for tabetics, but one that it is very hard for them to do well, is while standing on their feet to gradually bend their knees and bring their body towards the floor. They must be taught to do it not with the little starts and stops with which they always do it at first, but by a continuous movement. It is difficult but extremely useful, for it gives them confidence in their powers of erection and keeps them from "falling all of a heap" when they make a misstep or when suddenly surprised on the streets, or when they make a slight trip on the stairs, all of which accidents are characteristic and seriously discomfiting to the tabetic.

Then there is the bedridden tabetic, whom we must again divide into two classes,—the active one and the patient in whom long inaction or some complication has led to atrophy of muscles. For the first, in whom only incoördination and not muscular power is disturbed, the matter is comparatively simple. He is made to execute exact movements, as in the other case; to place the heel of one foot on various parts of the other leg, first under the direction of his eyes and then with eyes closed; to replace the feet in a given position after they have been displaced, and the like. A series of four movements executed at the word of command while an attendant counts, or to the ticks of a metronome, is very useful: 1, raise the leg; 2, bend at the knee; 3, stretch out leg, and 4, replace as before.

Then the patient may be placed in a sitting position on the side of the bed and directed to lift his heels from the floor and to place his feet in various positions. Then, firmly supported, he is put on his feet and movements attempted. After a while a go-cart commends itself for such patients. In the mean time they may practise touching with their feet various parts of the foot of the bed, or the

rungs of a chair or of a step-ladder put at the foot of the bed, until they learn the sense of position of their limbs once more.

As for the bedridden with atrophic muscles certain accessories are necessary. If you come with me to the ward I shall show you our three methods of compensating for muscle weakness here. They are not tabetic patients, but the principle is the same. Here is a boy with infantile palsy, anterior poliomyelitis, whose arms also are affected. Here, as you see, by a simple system of weights and pulleys, we overcome the influence of gravity on his legs, and so he can move and exercise them. His power over them has increased markedly as the result of the exercise, and you can see how proud he is to be able to move even thus. In the early morning before the weights are put on he can now move a little without his weights,¹ something he can never remember to have been able to do before. The encouragement afforded by this improvement in motion has caused a most healthy reaction that makes his general condition better than it has ever been.

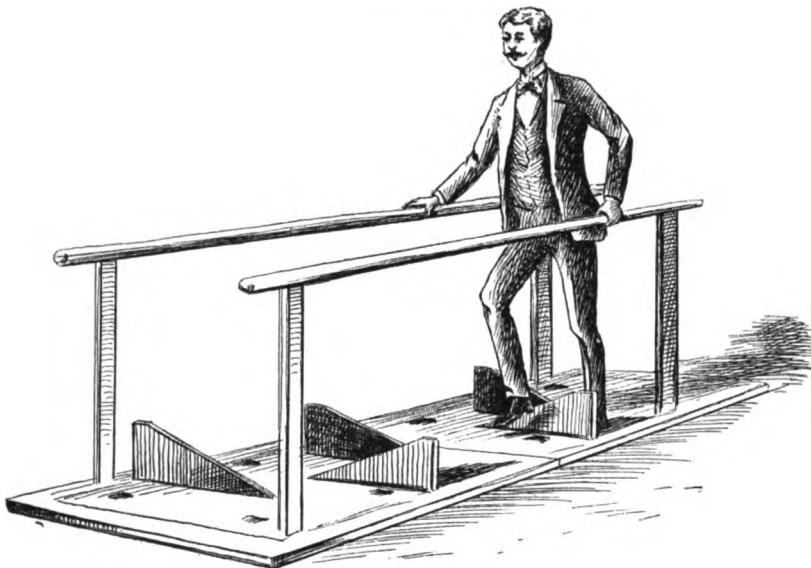
Our next case here is a neuritic paraplegia with atrophy; here the arms are unaffected and the system of ropes and pulleys enables his arms to help his legs. He, too, is improving, and is able to give himself exercise whenever he wishes without having to call an attendant.

Our third case, a man of thirty-five, is triplegic, both arms and his right leg being affected. There is syphilis in the history, and the symptom complex points to multiple gummatæ,—one near the motor centre in the left cerebral hemisphere, another in the cervical region of the cord, and, as he has the so-called saddle anæsthesia,—*i.e.*, of the neighborhood of the anus, the scrotum, and the inner sides of the thighs,—we conclude there is a gumma also in the conus medullaris. Here the process is gradually going back under specific treatment, but he is regaining the movement of his limbs very slowly. We have him put in a bath, and you see he moves his leg easily. Three or four times a day for some minutes he is put in a bath this way and allowed to exercise. The muscles regain their function much sooner than if they were exercised only by what movements he is able to perform against the dead weight of his leg. The bath itself is an excellent thing, for it keeps the patients clean and gives them something to look forward to.

¹ Two months later. This patient is now able to walk a little.

All of these methods could be used in helpless tabetic patients, as

FIG. 1.



Parallel bars and walking track with triangular obstacles to teach tabetics coördination and exact movements in walking.

FIG. 2.



Step-ladder, on the rungs of which or between them a patient may put his feet while lying in bed, and so practise exact foot motions.

you can easily see, and I have often had encouraging results from them. Now, as to the use of apparatus in the exercises. I have

purposely avoided mentioning them till now because the movement therapy can be carried on without them; but here are some that I have found of use because they occupy the patient's attention, interest and at times amuse him, and so induce him to be faithful in the exercises.

I have not spoken of the movement therapy for incoördination of the hands, because it is not so frequent or so bothersome a symptom. Where it occurs, however, it may be obviated in good part by exercises, as carrying the finger along the edge of a book or table, tracing the lines on a target with the finger, or tracing different geometrical figures with a pencil, following lines and spirals. This last will often make the handwriting more legible. In general, any acute exacerbation of symptoms during the course of the exercises should cause their interruption for a time. They may be resumed as soon as the general condition improves to any degree.

Now, as to contraindications to the exercises, there would seem to be only the one, that at times the pains become more frequent, when the exercises must be limited or stopped. I cannot help but think that in these cases they have been overdone. Fränkel, the inventor of the movement therapy, has also found that where there were frequent bladder crises these seemed to be made worse.

In the preataxic stage of tabes, when the pupillary knee reflex and perhaps the Romberg symptom are present, and yet no trouble of incoördination, a certain prophylaxis of the ataxia can be secured by the exercises, so that the ataxia as it advances is compensated for by the attention to the movements and a real state of incoördination never develops. I have seen some military men thus preserved from any external manifestation of their disease for two, three, or four years, and consider the prophylactic exercises of the greatest service. On the whole we have in this compensatory movement theory a distinct advance in the therapeutics of nervous diseases, which can be of the greatest assistance to our patients and the greatest consolation to us, when in the presence of a hopelessly incurable nervous affection.

TREATMENT OF CHOREA.

BY S. D. HOPKINS, M.D.,

Associate Professor of Neurology in Gross Medical College, and Neurologist to the Arapahoe County Hospital and St. Anthony's Hospital, Denver, Colorado.

IN no other disease is there such a wide diversity of opinion in regard to the use of drugs as in the treatment of acute chorea of Sydenham. All the sedatives and antispasmodics have been recommended in the treatment of this disease, but the majority of general practitioners are usually content with the regulation arsenic and iron medication. In my experience and practice the best results are obtained by the use of antipyrin, which was first called to the attention of the medical profession by Professor H. C. Wood, of Philadelphia.

In the last few years I have administered the drug by the method suggested by Dr. J. T. Eskridge, of Denver, Colo., which will be taken up in detail later in the paper. I am of the opinion that the use of this drug materially shortens the duration of the disease; although the majority of authorities agree that mild acute Sydenham's chorea lasts from six to ten weeks, while Gowers is of the opinion that it lasts from six weeks to six months.

In a tabulated view of nineteen cases¹ treated with antipyrin the duration is as follows:

Three mild cases, duration three weeks.

Seven mild cases, duration two weeks.

Five very severe cases, duration five weeks.

Two severe cases, duration four weeks.

Two severe cases, duration three weeks.

In this report of nineteen cases it will be seen that the duration of the most severe cases was only five weeks.

For a number of years I have refused to assume charge of a case of this disease if the parents and friends will not consent to follow my instructions. In the very mildest cases, in which there is

¹ Sixteen cases tabulated by Dr. Eskridge; three cases of my own.

only an occasional grimace of the face, or an infrequent twitch to the muscles of the hand or foot, I allow the patient to sit up a part of the day, and spend the remainder of the waking hours on a lounge. No violent exciting exercise, such as romping, running, etc., is allowed. The patient is placed on a nutritious, digestible, but non-stimulating diet. The stomach and bowels are kept in as good a condition as possible. The patient, to begin with, receives as many grains of antipyrin at bedtime as he is years old, and the dose is increased one grain each night until all twitching stops. At the beginning of the treatment of these mild cases I commence with one drop of Fowler's solution after each meal, and increase the dose one drop each day until the point of tolerance is reached; then the arsenic is discontinued for two or three days, or until all unpleasant effects of its administration have passed away, when the drug is again resumed at the dose reached when it was stopped. The dose is again increased one drop each day until tolerance is reached, when it is discontinued, and resumed after two or three days as before. As soon as the twitching ceases, the antipyrin at bedtime is discontinued, and the patient is given syrup of the iodide of iron after each meal, in from three- to ten-drop doses, depending upon the age of the patient. The arsenic and iron are continued for two or three weeks after all symptoms of the disease have disappeared and the patient has regained considerable flesh.¹

In all except the mildest cases, to which reference has just been made, absolute rest in bed, day and night, is insisted upon from the first. If I had to rely upon one method of treatment in the management of chorea to the exclusion of all others, I should unhesitatingly choose absolute rest in bed. When the little patient is placed in bed to begin its treatment (we will suppose it to be a child of a few years, or not more than fifteen years of age), I am in the habit of ordering as many grains of antipyrin, three times daily, after taking food, as the child is years old, and increase the dose one grain each day until all violent movements stop, when I begin with one drop of Fowler's solution after each meal, well diluted in water, and increase the dose one drop each day in the manner described. About the second or third day after the arsenical treatment has been added, the antipyrin is given only once each twenty-four hours, and the time for its ad-

¹ Medical News, September 30, 1893.

ministration is usually about eight or nine o'clock in the evening, thus securing a comfortable night for the little patient. After all but the most occasional twitching has stopped the antipyrin is discontinued, and syrup of the iodide of iron is given in connection with arsenic. It seems a little heroic to give a child of seven years of age from ten to fifteen grains of antipyrin three times daily, yet I have given a child of eight years twenty grains of antipyrin, three times daily, without the slightest apparent depression or untoward effect.

In using antipyrin certain precautions have to be borne in mind: if the patient has cardiac dilatation or a valvular lesion, antipyrin should not be given, although in the latter condition I usually substitute phenacetine combined with caffeine citrate, with very gratifying results. When temperature is present, the effects of antipyrin are so depressing that I use chloral, but not in as large doses as the former. While antipyrin is being given the patient must be kept in bed; for if the child is allowed to run around great depression is apt to follow.

After the severe twitching has ceased the case is put on Fowler's solution, beginning with one drop three times a day, and increased one drop each day until the point of tolerance is reached or until it has been taken for one week; it is then discontinued for three days, or until all unpleasant effects have passed away, when the same course of treatment is resumed. The dosage of Fowler's solution is held steadily at the point where all twitching ceased for ten days, after which it is decreased one drop daily. Along with the arsenic syrup of iodide of iron should be given, and kept up for a number of weeks after all twitching has ceased.

I wish to report a few violent cases treated with antipyrin and arsenic.¹

CASE I.—Rose K., fourteen years old, of Austria, has been troubled with violent choreic movements (bilateral) for six months. She has been under treatment a portion of the time. The jerking of the limbs is so violent that it is difficult to keep her in bed. She is placed on fourteen grains of antipyrin, three times daily, increased one grain each day. In four days, or by the time that eighteen grains of antipyrin are being given, thrice daily, all violent muscular twitching is stopped. She is now given one drop of Fowler's solution, thrice

¹ Cases I., II., and III. occurred in Dr. Eskridge's practice.

daily, and the dose is increased one drop each day, and the antipyrin is reduced to one dose of eighteen grains daily. In the mean time absolute rest in bed is insisted upon. At the end of the second week the choreic movements have nearly ceased, and by the end of the fourth week she is discharged, cured.

CASE II.—Helma B., thirteen years of age, has been suffering with violent choreic movements of the left side for three months. During this time she has been under treatment, but has been allowed to be up and walk around all day long. When she entered the hospital the twitching was violent. She is placed upon large and increasing doses of antipyrin and kept at rest in bed. In a week's time all violent movements have ceased. At the end of eighteen days she is now practically cured, under the antipyrin and arsenic treatment.

CASE III.—Charles R., fourteen years old, was admitted into the nervous ward of the County Hospital about two weeks ago. There was violent jerking of the muscles of all the limbs and of the face. He had to be strapped in bed, as the irregular muscular action made it impossible for him, unassisted, to remain in bed. He states that he had been in this condition for six months. Articulation and deglutition are difficult. He is placed upon the same treatment given in Cases I. and II., with equally good results.

CASE IV.—Arthur H., aged four, always enjoyed good health until present illness began. In first part of April, 1899, his parents noticed that he was very irritable; and in a few days he began to twitch (bilateral), and gradually increased until at the end of a week he was unable to perform any voluntary movements. Experienced great difficulty in swallowing, and was unable to make an articulate sound which could be recognized as speech, nor could he sit up in bed without assistance. No temperature or cardiac lesion. He was put to bed; given four grains of antipyrin, three times a day, and increased one grain daily, until eighteen grains were reached, when the violent jerks stopped; then the arsenic and iodide of iron were begun, and the case discharged, cured, in five weeks.

CASE V.—Carrie K., aged ten, has had bilateral twitching for the past three months. For the last two weeks the muscular movements have become very violent. No heart lesions or temperature. Was put on ten grains of antipyrin, increased one grain daily; when sixteen grains were reached violent twitchings ceased. The subse-

quent treatment was the same as in the previous cases. Discharged, cured, in five weeks.

CASE VI.—Katie E., aged thirteen, has suffered from choreic movements for the last eight months. At the time of my first visit all the muscles of the body were affected, and the twitching so violent that she had to lie tied in bed. Articulation and deglutition markedly affected. No fever. Mitral regurgitant murmur present. She was put on phenacetine, grains thirteen, and one grain of caffeine; the latter not increased daily. Twenty grains were reached before violent twitching ceased. Subsequent treatment the same as previous cases. Discharged, cured, in four weeks.

THE SURGICAL TREATMENT OF BENIGN AND MALIGNANT STRICTURES OF THE CESOPHAGUS.¹

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF ROSTOCK, GERMANY.

BY PROFESSOR GARRÈ,

Director of the Surgical Clinic and Professor of Surgery at the University.

GENTLEMEN,—Our first patient this morning is this little girl, whom you have seen before, as she was with us some months ago. The history of her complaint is, briefly, as follows: In June, 1897, she swallowed a certain amount of lye,—i.e., she took some solution of caustic soda into her mouth by mistake; as it burned her tongue she made an almost involuntary effort to swallow, and the evil was done. How much lye there was is not very certain; there does not seem to have been very much, and the mother thinks there was scarcely more than a few drops. How concentrated it was and how much ill it worked you will be able to judge of from the story of the case subsequently.

Since then she has been able to swallow nothing but liquids. There have been times when she seemed some better, and then she would grow worse again. She was treated by dilatation with bougies at a neighboring university for some months, and was able to swallow so well, and picked up in health and strength so much, that she neglected the warning that she must never let a day pass without the passage of a bougie. Almost imperceptibly the old condition reasserted itself until, about ten days ago, she became absolutely unable to swallow anything, even liquids. Under these circumstances she was brought to us. As might be expected, she was in a most miserable condition, unable to take food, rapidly emaciating, and suffering torments from thirst, which the taking of liquid into her mouth and endeavoring to swallow it scarcely seemed to do anything for. Rectal feeding, and especially the plentiful injection of

¹ Reported by James J. Walsh, Ph.D., M.D.

liquids, at once made her condition more tolerable, and now we must consider what we are to do.

The mode of origin of the stricture gives us a good idea of its form and of its character. The walls of the oesophagus a little distance below the trachea lie normally in contact, so that the effect of a corrosive liquid is exerted upon its entire circumference when it gets to this point. The result of the corrosive is necrosis of the cells. If the corrosive liquid is small in amount, or is only weak, then only the superficial cells are destroyed, and a lasting effect may not be produced. Unfortunately, the liquid swallowed is only, too often, strong enough to cause not only necrosis of the epithelium, but also of a layer of the submucous tissue.

The first symptoms are always burning pain, usually followed by vomiting. This latter is a very fortunate protective reaction of nature, by which the offending material is gotten rid of and further destruction of tissue prevented. Shortly afterwards some blood-stained mucus is apt to be coughed up, and then shreds that resemble diphtheria membrane and are of a similar character, since they consist of patches of necrosed mucous membrane. Where the tissue destruction has taken place ulcers develop, and these during the process of cicatrization cause contraction of the tissues and the stricture.

As the result of the inflammatory reaction just after the accident, there is a swelling of oesophageal tissues and a hyperæmia which gives rise to a painful sense of pressure and constriction in the throat and prevents swallowing. When the necrosed parts are thrown off after a day or two, and ulcers develop, there is relief afforded from these symptoms, which is extremely encouraging to the patient, and, by permitting at least liquid food to pass, makes his condition much more tolerable. This sense of relief does not last long; the ulcers become painful and irritable from the passage of even liquid food, and as they heal stricture of the oesophagus gradually develops from cicatricial contraction.

Notwithstanding the seeming ease of diagnosis of cases of acute stenosis of the oesophagus by traumatic or corrosive action, there are times when a doubt may arise. The swallowing of a corrosive liquid happens so often in children who can either tell nothing of the circumstances, or whose admissions under the excited questioning of parents may be anything but trustworthy, that at times you may

have to recall certain differential diagnostic points. Mere pain and difficulty or impossibility of swallowing are not enough to make a diagnosis. Children may have an ordinary sore throat and refuse to swallow because of the pain, or paralysis of the soft palate may occur and liquids find their way out through the nose, and parents find in some incidental circumstance grounds for the suspicion that a corrosive liquid has been taken.

The important thing to look for in all such cases is traces of the corrosive action of the liquid in the mouth and upper digestive tract. It is often surprising, however, how much severely corrosive liquid may be taken at a gulp and swallowed before the realization of its nature has occurred. Several ounces of carbolic acid or of concentrated lye may be swallowed thus, and surprisingly large quantities of even the stronger mineral acids have been found in the stomach after death, having been swallowed inadvertently,—*i.e.*, before enough reaction had been produced in the mouth to cause their rejection. Sometimes this is doubtless due to the fact that the inevitable tendency, or, at least, habitual, almost instinctive, action, is to swallow something that is easily swallowed, if it produces irritation. Another and more important reason is that the reflex motions of deglutition are accomplished before the nervous reflexes of taste and irritation are carried to the consciousness.

As the time is so short, then, in which the liquid stays in the mouth, the changes sometimes found here are not at all correlative to the amount or strength of the liquid that has been taken, and too much weight must not be laid on the extent of the changes that are to be noted here. Much more important is the fact that there are distinct traces of corrosion, showing that a strong etching fluid has been swallowed. Of course, in most cases where strong corrosive liquids are swallowed, the necrosis of oral and labial mucous membrane is so outspoken as to leave no doubt of what has happened.

Later in the case the symptoms of œsophageal stricture are very typical. Our patient here, for instance, is able to take a certain amount of milk, but after two or three small swallows is absolutely unable to get any more down. The œsophagus has filled up, and will hold no more. After a moment or two, with a sort of hiccough, a certain amount of it is returned. I say returned, because it is not vomited; there is no action of diaphragm or abdominal muscles in its regurgitation. (Demonstration.) When she takes small quanti-

ties and then makes a series of successive swallowing efforts, she thinks she succeeds in getting some of it down. When I listen over the course of her œsophagus behind for the series of sounds characteristic of swallowing, I do not catch them. If there is a small opening through the stricture, it is of a tortuous character and very narrow, and the liquid finds its way through it only in extremely small quantities and very gradually, and without producing the characteristic swallowing murmur.

She is, however, able to take more liquid than would be contained in an œsophagus of normal calibre closed at the point of the stricture here, and after the accumulation of liquid she sometimes regurgitates a considerable quantity. It is probable, then, that here, as in many cases of stricture of the œsophagus, under the constant pressure of even the slight column of liquid that the œsophagus will hold, there has been a dilatation of the œsophagus just above the stricture. This would help to account, too, for the ease with which regurgitation takes place, for wherever muscular tissue under conditions of reasonably good nutrition is put on the stretch, hypertrophy of the muscle fibres occurs. This is strikingly exemplified in the heart and in the muscular coat of the intestines just above a stenosis. The muscularis of the œsophagus, then, has overgrown during the dilatation, and so easily contracts enough to cause regurgitation with scarcely an effort.

In the dilated portion of the œsophagus, called technically an œsophageal diverticulum, a certain amount of swallowed material may remain for some time. When we first had our little patient try to swallow some milk this morning, as you saw, after taking scarcely more than a sip of a couple of spoonfuls, she regurgitated a couple of ounces. It had evidently been in the œsophageal pouch since last evening. That it had not been in the stomach was clear from the little alteration it had undergone. It was not coagulated, it had none of the fatty acid smell of stomach contents that have remained in the stomach for hours, and was only mingled with a certain amount of mucus.

We have a patient, then, to treat who seems to have an impermeable stricture of the œsophagus,—at least, for food. We shall find its position and further test its permeability by sounds. Of course, I need not insist with you—I have often done it before—on the necessity of being sure that an aneurism is not present in any case

of difficulty of swallowing before attempting to pass a sound. Here such a precaution is obviously less needed, yet a careful physical examination of the thoracic organs is important to be sure that normal anatomical conditions may be expected.

A medium-sized sound meets with an obstacle about seventeen centimetres from the teeth. A small one meets an obstacle at the same point. At times a smaller sound will be found to go farther than a larger one, showing that the structure is at its upper end funnel-shaped. This result is a little more encouraging than the one we have found here, since in a funnel-shaped stricture some of the smaller sounds will find a passage, by gentle manipulation, if one of sufficient size exists. After all, it is to be remembered that besides the stricture a certain amount of spasm may exist from irritation, and if the end of the sound be once engaged in the passage through the stricture, the spasm may be overcome.

We have patiently tried to secure the passage of small whalebone and filiform sounds through this stricture and failed. Of course, we employed no force. It was the custom to say that in these cicatricial strictures a certain amount of force might be safely used to secure the passage of a sound, where an experienced hand seemed to detect by the feel that the end of the sound was engaged in the stricture. Of course, it was never considered allowable to employ any force in malignant strictures, as the succulent neoplastic tissue yielded too easily and permitted the sound to tear its way into the pleura or mediastinum. Of late it has come to be recognized universally by surgeons, from sad experience on the part of many of them, that force must not be employed in any stricture of the oesophagus.

When we found that the smaller bougies would not pass we made use of an expedient first employed by von Hacker, of Innsbruck, and adapted from a corresponding proceeding employed in strictures of the urethra. We passed a tube, open at both ends, and through it a series of filiform bougies. The tube prevents folds of the oesophagus from getting in the way, and each of the filiforms by its presence tends to dilate the oesophageal tube just above the stricture, and so permits one of them to find its way into the narrowed passage. If one passes, it is allowed to remain for some hours, by which time it will usually, by the relaxation of spasm and the pressure absorption that it occasions, have made room for another beside it. The two

may be left for twenty-four hours, when a small bougie may often be passed and then further sounding becomes impossible.

We were unable to pass a filiform in this manner, so we tried the method suggested not long ago by Socin. A small spherical piece of tin—since this metal is not acted upon by the digestive secretions—is fastened to a silk thread and given to the patient to swallow. Sometimes the small body finds its way through the stricture after swallowing efforts, when the thread is allowed to remain for some time. Its presence acts like a filiform to produce a certain regression or softening of tissues around it, so that beside or along it as a guide, but, it is to be always understood, without the employment of any force, a filiform or a small bougie may find its way. This method also failed here, though tried faithfully two or three times. Naturally all these attempts were not made one right after another, for the irritation of the upper end of the stricture would in that case produce so much hyperæmia that the stricture would be tighter than ever. Twenty-four or forty-eight hours were allowed to elapse between each attempt. In the mean time the patient's general condition is much better than it was, as she is being fed carefully by the rectum, and especially is absorbing plenty of liquid, for she was in a most pitiable state from protracted thirst. She stands rectal feeding very well.

So far, then, we have failed, and have a stricture absolutely impermeable for instruments. Had we succeeded in passing the stricture with a bougie, we would have allowed it to remain for from fifteen minutes to half an hour, not longer, though some surgeons have spoken of leaving it for hours, but it does not seem advisable. The trophic condition of such cicatricial tissue is not of the best; the pressure of a bougie may then lead to decubitus, or, at least, to a lesion of the mucous membrane of the cesophagus, and, as pathogenic bacteria from the mouth are continually passing down with the saliva, infection may easily take place, and infection, on account of the neighborhood of important and susceptible organs, is always serious here.

Each day a larger bougie would be used, until a passage the size of your index-finger at least is secured. This much can usually be accomplished in about six weeks, and this is as far as it is safe or advisable to go, and, besides, this suffices perfectly for nutrition. The passage of bougies must not stop here, but must be continued daily or

at least every two or three days for a long time, perhaps for life, so as to overcome the contractile tendency of the cicatricial tissue. This tendency can never be completely overcome, however, and the patients usually must learn to pass the bougies for themselves, which they commonly do with ease.

The most insistent warning of the danger of neglect will not be enough for some patients, and they will fail to pass the bougie regularly, the stricture contracts some, and they are unable to pass it, and then they let the matter run on until difficulty of deglutition develops once more. It is better, then, always to have such patients report at least every two months, in order to be sure that everything is going on all right.

In certain cases of obstinate and retractile strictures the procedure is not so simple. They refuse to take a larger sound from day to day, and are as bad the next day after the passage of a bougie as they were before. In such cases the expedient of using the dilating force of a rubber tube that has been stretched and so reduced in size, and then allowed to resume its original length, with a consequent increase in its thickness, may be employed. It was first used in the dilatation of urethral strictures, and was first employed by von Hacker in the oesophagus. Over a round-ended solid bougie a rubber tube closed at the end is drawn and then stretched. While the rubber tubing is stretched and thin the bougie thus prepared is passed into the oesophagus, and then the rubber is allowed to relax. The gradual dilatation thus effected is, as a rule, most successful.

But these procedures are for permeable strictures, while in our case so far we seem to have an *impermeable* one. If, after another trial, this time during narcosis, we fail to pass a bougie, then we shall have to think of a gastrostomy; for, while our patient has picked up during rectal alimentation, that is only as compared to her former miserable condition. Feeding per rectum never suffices for any length of time to keep up the balance of metabolism. Unless we would see our patient decline into a condition that would render an operation dangerous, we must find a way to gastric nutrition.

A gastrostomy will accomplish this primarily, and then we can try a *retrograde dilatation of the stricture*. This is often a much easier procedure than the direct dilatation from the mouth. The corrosive liquid usually produces its worst effect at the upper part of

the oesophagus, so that from below one may find a more or less funnel-shaped route leading to the point of greatest resistance.

Then, if the stricture is low down in the oesophagus, the finger may be inserted from the gastric end as far as the stricture, and will then serve as a sort of guide to a sound introduced from the mouth. Or, after a few days of gastric feeding, and absolute absence of all irritation from attempts to swallow food or the passage of bougies, the stricture may relax a little, when the tin bullet with the silk string may be successfully swallowed, and being brought out through the gastric fistula prove a director for further manipulation. On this guide, then, a series of rubber tubes may be pulled gently through the stricture, at first singly, then one within the other, until dilatation with sounds may be taken up and the gastric fistula allowed to close, as it will of itself in children; or its edges may be freshened and brought together with some silk sutures in adults.

Other expedients may be employed. For instance, after the silk string has been gotten through the stricture and caught through the fistula from below, a sound with an eye in it, somewhat like the tunneled urethral sounds or catheters, may be threaded onto it and pushed on into the stomach.

Gussenbauer has recently introduced a combination of oesophagotomy and gastrostomy that may be a very useful procedure. Where the manipulations so far mentioned fail to enable the surgeon to pass the stricture with a sound, an oesophagotomy, by bringing him closer to the stricture, may enable him to pass a filiform, practically under the direct guidance of sight, or even a small sound, and thus further dilatation become possible.

There are certain cases of stricture of the oesophagus in which the oesophagoscope may be of service. With it we are enabled to see the upper surface of the stricture and to get an idea of its form and of the possibility of a channel in it much better than with the sound. At times a practised hand may, under the direct guidance of the eye, pass a sound where the tactile sensations were not sufficient to enable us to find the passage. This is especially true where folds or pseudo-ventricles exist just above the stricture. I have here for your inspection an oesophagoscope and some of the plates that Professor von Hacker, of Innsbruck, has made for various conditions, especially the presence of foreign bodies in the oesophagus.

We shall after the clinic have our little patient anesthetized, and

shall make a last careful attempt to pass a bougie. If the attempt is not successful, and I fear it will not be, I shall at once proceed to do a gastrostomy, and after a day or two will attempt the retrograde dilatation of the œsophageal stricture.

Our next patient this morning is a very different case from the one we have just dismissed. She is about to leave the hospital now in a very satisfactory condition, and so I present her. She is a young woman of twenty-eight, who for several weeks before her confinement, in September, suffered from difficulty in swallowing, so that she was able to take only soft food. After her confinement swallowing became even more difficult, so that she could after a week or two take only liquid diet, and that with the greatest effort. She emaciated rapidly, and came to the hospital in a very weak condition.

Examination showed the presence of an obstacle to the œsophageal sound twelve centimetres from the teeth, at the beginning of the œsophagus, therefore. With the finger and counterpressure upon the larynx, we were able to feel, just behind the larynx, a mass of hard, resistant tissue at the beginning of the œsophagus. With the laryngoscope there was no tumor to be seen; the arytenoid cartilages were somewhat swollen; external palpation gave no result.

The diagnosis was carcinoma of the upper part of the œsophagus. The condition of the patient was so bad that a radical operation, it was feared, would prove fatal. An œsophagotomy was done on the left side of the neck as low down as possible. After the opening of the œsophagus a hard tumor was to be felt above, and a small portion of it was excised for microscopical examination. It proved to be carcinomatous. Our patient began to pick up at once after the operation, so that three weeks later we felt justified in proceeding to attempt radical extirpation.

We found, on attempting to separate the œsophagus and the trachea, that the latter was infiltrated by the carcinomatous growth, so we took away with the resected œsophagus the first five tracheal rings, obliterating afterwards by stitches the lumen of the trachea just above the tracheotomy canula. The right lobe of the thyroid was also found to be infiltrated, and was therefore removed. As it would have been impossible to make up for this large defect in the trachea, we preferred to permanently interrupt its continuity completely, and as this would leave the larynx as an utterly useless organ, we removed its cartilages and made use of the mucous membrane,

whose vascular connections, especially from above, were carefully preserved, to replace the mucous membrane that had necessarily to be removed from the oesophagus.

The mucous membrane of the arytenoid cartilages was attached to the lower end of the resected mucous membrane of the pharynx, the subglottic mucous membrane was split in the median line in front, up to the base of the epiglottis, to simplify the operation, and then fastened to the upper end of the resected oesophageal mucous membrane below, and the arytenoid mucous membrane, that already had its place in the plastic work, above. A tampon was inserted to prevent the flow of saliva from the mouth over the plastic tissue.

The patient stood the operation very well. For the first eight days afterwards there was considerable disturbance of temperature, due to the formation of an abscess that by gravity began to find a way into the mediastinum, but by carefully lifting the foot of the bed, allowing no pillow, making every avenue possible open for drainage, and changing the bandages several times a day, this danger was overcome and the further progress of the case was almost without incident. On the tenth day some of the stitches in the displaced laryngeal mucous membrane gave way and the defect was filled by transplantation.

The patient gained rapidly in weight and in her general condition. Three weeks ago we were in a position to close the oesophageal chink that remained by small double-door flaps over which the external skin could be brought together without further trouble. For two weeks after this a small fistula remained just above the jugulum, but this now has closed, and she is able to swallow soft food without any difficulty.

She is now practically well, and she has already learned, by means of a whispering voice, to make herself passably understood.

It was a serious question to my mind how the delicate sensitive mucous membrane of the larynx would behave under the circumstances. Its innervation remains the same as before, for, in order to avoid trophic troubles, we spared the nerves leading to it as much as possible,—the superior laryngeal nerve was not injured. As this mucous membrane is, in its original location, extremely sensitive to the slightest irritation, it remained to be seen how it would take up its new duties in the oesophagus where it would be necessarily subject to frequent, in fact, almost constant, irritation. It was to be feared

that the tendency to cough would disturb the taking of food so much as to perhaps render it impossible. Our forebodings were not justified, however, and so far it allows ordinary soft food to pass over it without undue reflex irritation. While swallowing is normal, however, the attempt to pass a sound is followed, as you see, by an immediate paroxysm of coughing.

I think that we have succeeded in this case in making a total extirpation of the neoplasm, and I confidently look for a continuance of the present condition without relapse.

Our two cases of to-day show the different methods of treatment of the malignant and the benignant strictures of the œsophagus. For the malignant strictures the only hope of anything like an enduring relief to the patient is the complete extirpation of the growth. Unfortunately the tumor is so situated usually that this is impossible. In these favorable cases of carcinoma high up in the œsophagus, the operation is technically so difficult, and the filling up of the œsophageal defect by plastic material afterwards attended with so much trouble, that the operation has been a most unsatisfactory one. This is the first time that the laryngeal mucous membrane has been used successfully for plastic repair of the œsophageal defect, and I think that the case will prove a suggestive example to others, of methods of procedure for these extremely difficult cases, each of which will have to be a subject of special study by itself.

REMARKS UPON THE TREATMENT OF DIPHTHERIA, WITH ESPECIAL REFERENCE TO THE TECHNIQUE OF INTUBATION.

BY SAMUEL G. DABNEY, M.D.,

Professor of Physiology and Clinical Lecturer on Diseases of the Eye, Ear, Nose, and Throat in the Hospital College of Medicine, etc., Louisville, Kentucky.

A few practical remarks on intubation for diphtheria or membranous croup, with especial reference to the technique of the operation, may not be uninteresting.

First. In my judgment membranous croup and laryngeal diphtheria are one and the same thing. This opinion is based upon the fact that the microscope shows that in at least twenty-eight out of every thirty cases the Klebs-Loeffler bacillus is found in the membrane of the so-called membranous croup.

Second. This conclusion is reached from the fact that the so-called membranous croup is in many cases—and I have seen several such instances—preceded by disease of the throat; there has been a little deposit of membrane on the lower part of the tonsil, perhaps, which has been overlooked by the physician. This membrane may have existed for some time and escaped detection.

Third. From the fact that the manner and clinical course of membranous croup are the same as in diphtheria.

It was formerly thought that these two diseases were entirely separate and distinct, chiefly for these reasons,—viz., that diphtheria is accompanied by enlarged glands in the neck, and also marked systemic symptoms, whereas in true membranous croup, as formerly called, those two symptoms are lacking, or, if not entirely lacking, they are present in very slight degree. The reason for this is easily seen: As long as the membrane is confined within the larynx the absorption from this membrane is slow, because the lymphatic distribution of the upper part of the larynx is not very abundant; so involvement of the lymphatic glands of the neck and marked constitutional

depression are not apt to occur so long as the diphtheria is confined to that place.

I conclude, then, that acute membranous inflammations of the larynx, leaving out traumatism, injuries by burning steam, etc., are all diphtheria. This opinion may be relied upon as being correct, and in New York and other cities to-day they require the same isolation in cases that were formerly called membranous croup as they do in diphtheria, and such cases are reported as diphtheria chiefly on the basis of bacteriological examination. The cause of diphtheria is conceded to be the Klebs-Loeffler bacillus.

This paper is intended to deal especially with the indications for intubation, how the operation should be performed, and what treatment must be instituted before and after the operation. If the physician is called to a patient with membrane in the throat, his first thought nowadays should be the use of antitoxin, if there is even a probability of its being diphtheria. He should not wait to have an examination made by the microscopist. The probabilities are that to a majority of average practitioners a microscopical examination will be impossible, unless the physician is competent to make it himself: and, with all due respect to his microscopic teaching and his instruction in bacteriological work, I question whether he will be very expert. He must trust, then, to his clinical diagnosis in these membranous cases rather than to the microscope. Therefore, if he is called to see a case that is probably diphtheria, I advise that antitoxin be used promptly. It has been used in hundreds of thousands of cases without bad effect. In one mass of statistics where antitoxin was used for prophylactic purposes in healthy children, there was not a single serious consequence in fifteen thousand cases, which is strong evidence that antitoxin itself is a harmless agent. The physician need not hesitate, therefore, to make use of it without fear of harm resulting therefrom. There are many reliable preparations; four or five, perhaps, of the leading preparations on the market are equally reliable. There has been one marked improvement in it within the last year or two, and that is, they have made the antitoxin more concentrated, and the same effect may be obtained with much less bulk than formerly, so that nowadays about five cubic centimetres are enough for one injection in the most malignant case of diphtheria. There are several favorite points for its injection; personally I inject it into the outer part of the thigh. I usually ask upon which side the

patient is in the habit of lying, and inject in the opposite side. Some prefer the gluteal region, others the interscapular spaces, still others the loose tissue of the abdominal walls; it really does not make a great deal of difference which point is selected. When antitoxin is injected, the spot selected must be thoroughly cleansed with soap and water, and some authorities also advise thoroughly sponging the part with a solution of bichloride of mercury one to two thousand, but I do not attach much importance to this. Antitoxin should not be used from bottles that have been left open; it should be a freshly opened bottle for each occasion.

Having thoroughly scrubbed the point selected for the injection, of course the operator having first of all cleansed his hands with soap and water and a nail brush, he will simply draw up into the syringe so many cubic centimetres of antitoxin, insert the needle, and deposit the contents of the syringe immediately under the skin. The amount injected will depend upon the concentration. In an infant under one year, one thousand units may be used; in a child over two years of age, fifteen hundred units. Units in this connection have reference to the concentration of the serum; consequently in severe cases in children over two years of age, particularly where there is involvement of the larynx, an antitoxin should be procured containing fifteen hundred units, and two thousand units would be better if there is laryngeal obstruction. Having poured or drawn the proper volume of antitoxin into the syringe, the needle is inserted, the operator taking up a little piece of the skin just as he would in giving a hypodermic injection, and the antitoxin is deposited just beneath the skin. The site of the injection may then be covered with a small piece of absorbent cotton, using some simple antiseptic preparation if desired, to keep it clean and to keep the part closed and protected. I have never seen an abscess following the injection of antitoxin, and I have used it in a great many cases, nor have I seen any considerable swelling or soreness following such an injection.

Improvement may be expected in from twelve to twenty-four hours. Improvement consists in limitation to extension of the membrane. The first thing, I should say, would be a cessation in formation of the membrane, it comes to a standstill; second, the membrane disappears sooner; third, the constitutional symptoms abate, the child improves in color, pulse, and even in its breathing.

The remedies in which I have most confidence besides antitoxin

in the treatment of diphtheria are strychnine, mercury, and whiskey. I mention strychnine and mercury first because I think strychnine is the most serviceable stimulant and tonic that we have to sustain the action of the heart and prevent the depressing effects of the disease. It is astonishing what large doses young children will stand of this drug when they are under the depressing influences of such poison as diphtheria. The physician is more apt to give too little strychnine than too much in these cases. For a child five years of age, begin with one-fortieth of a grain every three or four hours, when the quantity may be increased as the occasion requires.

Mercury is best given in the form of calomel in frequently repeated small doses. It is desired to make the bowels act thoroughly and keep them open.

Of whiskey little need be said except that it is an old, time-honored remedy in the treatment of diphtheria. It is called the Brooklyn treatment, because it was inaugurated in that city during the terrible epidemic several years ago.

It is not my intention to dwell upon the internal treatment of diphtheria, and have only mentioned a few of the most practical points.

As to local treatment: It is amusing how widely different is the practical treatment of this disease in the hands of different members of the profession. Some prefer to use peroxide of hydrogen, some Monsell's solution, some bichloride solution, others carbolic acid and lime-water. Those who use these agents state that they do so for their antiseptic effect; they say that these agents make their way into the membrane and destroy the germ of the disease, in addition to their being cleansing agents. If one of these agents could be made strong enough in solution I have no doubt it would destroy the germs, but a stronger solution than it is possible to make would have to be used considering the time it could remain in contact with this surface, so that chiefly the mechanical cleansing effects only are obtained.

A year ago last summer I made a brief visit to Boston and New York, and visited the Boston City Hospital and the Willard-Parker Hospital of New York with a great deal of interest to myself. These are the two chief diphtheria districts in the United States; they had about thirty cases of diphtheria in the Willard-Parker Hospital the day I was there. All these cases received antitoxin. Their chief

internal treatment was strychnine. They also douched the throat with normal salt solution, using an ordinary fountain syringe. I remember making a short report of my visit before one of the Louisville medical societies after my return, and one of the physicians present made the statement that he did not see how they could douche a child's throat with a fountain syringe. It is done very easily. The child's tongue may be held down if desired, or the operator can simply carry in the rubber nozzle and press the tongue down with that. Of course the child must be lying on its side, and must also hold its breath for a moment. The water should be allowed to run freely over the surface of the throat, and it will wash off the membrane better than anything else. They used for this purpose a normal saline solution and a fountain syringe. Saline solution is about the best local treatment. However, there are some children who will not stand this method, and it is necessary to resort to the use of the spray.

To return to the question of laryngeal obstruction and how to treat it: Suppose the physician is called to see a little child, and no membrane has formed in the throat, or, at least, none has been seen; let it be supposed, further, that the family physician or the parents think the child has the croup. The child, perhaps, has a peculiar characteristic metallic cough, a cough which is hard and dry; the voice is either lost or very much impaired; not infrequently is quite lost; there is seldom any pain; the pulse is good in the early stage of the disease; the skin is of good color, and the child is breathing quietly and easily; there are no marked evidences of obstruction. In such a case, if there is membrane in the larynx antitoxin should be given at once. Nothing else need be used at that time, but the physician should see the child again in the course of a few hours. Suppose antitoxin for some reason is omitted, the membrane rapidly increases, obstruction becomes more marked, and all symptoms are aggravated. It will be found that the child is restless; it will toss from side to side, and its hands will be continually thrown up to its neck. If the covers be drawn down from the child's neck and chest, and the physician looks below and above the clavicles, he will observe, if there is marked laryngeal obstruction, a sinking of the space above the clavicles and beneath them with each inspiration. The spaces between the costal cartilages are sunken in instead of being expanded with each inspiration. When that stage is reached, if it is continuous and progressive, it is time to operate. The physician should not wait

until the lips become blue, until the restlessness is increased, until the child is actually struggling for breath. Humanity as well as science demands that the patient be given speedy relief.

The question is, How shall we operate? Fifteen years ago there would have been only one answer,—viz., open the windpipe, an operation that would hardly have been resorted to in the stage I have just described, as the family would certainly not permit it. It is a horrible thing to them to think of the windpipe being opened, to see the blood, and the tube sticking into the neck on the outside. The treatment nowadays is intubation. Twelve years ago a surgeon by the name of O'Dwyer, after two or three years of careful investigation upon animals and children, treating a large number of cases of obstructive diseases of the larynx, announced that he had invented a set of tubes with instruments for their introduction and extraction, for the relief of acute laryngeal obstruction. O'Dwyer announced his discovery, and the method came into general use after a few trials. The tube is inserted with the introducer, and by pressing the knob the obturator is released and withdrawn, leaving the tube *in situ*. The O'Dwyer case contains many tubes of various sizes. A tube suited to a child one year old would not be adapted to one of seven, etc., hence O'Dwyer devised a table and system of marking the tubes to indicate the size required for children of various ages. The tubes have a shoulder at the top which prevents their slipping down below the vocal cords; they have a little bulge in the centre which helps to steady them in place.

When the physician goes to put a tube in a child's throat, he should first of all fasten a string through the hole in the tube, upon which he can pull if occasion requires. Suppose he is called to see a little child lying in bed, breathing hard,—perhaps he has heard it breathe before entering the room,—and with a metallic cough (the latter may or may not be present); he pulls down the covers from its neck, and on each inspiration there is a sinking in of the supra- and infraclavicular spaces; farther down all the ribs are drawn in on inspiration. Even although there is no blueness of the lips, if the child is in great distress with the symptoms I have described, when they are progressive, the condition demands immediate relief. Have the child taken up by an assistant, a nurse, or the father or mother, have a towel pinned around its arms; then with the child seated in its father's lap, its head resting against its father's shoulder, have an

assistant stand behind both the father and child and hold the child's head; insert the gag on the left side between the back teeth, opening the mouth widely. A tube of proper size for the child's age with string attached should already have been prepared. Then the physician must go by the sense of touch; he must introduce his left forefinger into the child's mouth back until it reaches the epiglottis. When the epiglottis is felt and recognized by the sense of touch,—he may go a little farther if desired and feel the arytenoid cartilages,—it is drawn forward, with the palmer aspect of the left forefinger pressing the tongue downward; then guide the introducer along the under surface of the forefinger, introducing it first with the handle close to the child's chest; then, as soon as the larynx is reached, the handle is drawn up level with the child's mouth; then guide the tube with the left forefinger, and be sure that it enters the larynx, press the spring, withdraw the introducer, and leave the tube in. He can now, if desired, remove the gag and see if the child breathes quietly and easily, then should once more insert his finger and see that the tube is in place, pressing on the tube with a moderate degree of force to assure himself that it is in the larynx; if it is, then with scissors he may cut the string and pull it out.

The above, in short, is the technique of intubation of the larynx.

The mistake that is most apt to be made in attempting to introduce the tube is that it will go into the pharynx and oesophagus instead of the larynx; an inexperienced operator is almost sure to do this at first, as the work must be done largely by the sense of touch. By practising on a dog's larynx, for instance, one can get a very good idea of the manœuvres required in order to successfully perform the operation upon the living human subject. If the cadaver can also be practised upon, it would be advisable. In performing the operation, when it can be done, the head-mirror should be used; while it is true not much can be seen even with the aid of the mirror, still it is of assistance in guiding both the finger of the operator and the instrument, though, as already indicated, most of the work is done by the sense of touch. The chief thing is to educate the finger; the difficulty that will at first be encountered is in recognizing the epiglottis. In the child's throat the operator is working in a very small space. The first intubation attempted on the living subject will likely be performed with considerable difficulty. I know my first one was, and I was familiar with the throat, too. After the first half-dozen or so cases

the operation will be comparatively easy. In my opinion the difficulties have been greatly magnified even by those with large experience. O'Dwyer has written a book on the subject, which is a very excellent work, but I think he magnifies the difficulties attending the operation.

Getting the tube out is usually the more difficult procedure. A great deal depends upon the assistant in removing an intubation tube; the child's head should be held well up, and care must be taken in the introduction of the extractor; be sure that the blades of the extractor open the proper width; regulate the size of the opening to which the extractor opens by the dimensions of the tube. This is done by means of the screw at the bottom. The chief thing is to have the child's head held well up; the operator, sitting down in front of the child, will insert his left forefinger, pull the epiglottis forward, carry his extractor down, guiding it with his finger until it is felt to enter the tube, then press the handle and withdraw. The handle of the instrument should not be pressed until the operator is sure the extractor is in the tube. As a rule, the tube is worn from three days to a week. In young children it is better to allow the tube to remain not less than five days. The older the patient the sooner may the tube reasonably be taken out.

THE OPERATIVE TREATMENT OF HIGH MYOPIA.

BY EDWARD JACKSON, A.M., M.D.,

Of Denver, Colorado.

MYOPIA is generally due to elongation of the eyeball. This, occurring without change in the refracting power of the cornea and crystalline lens, causes the light from distant objects to be focussed in the vitreous before it reaches the retina; so that all distant objects are seen indistinctly, and the eye is "near-sighted." In a few cases the eyeball is not elongated, but the refracting surfaces of the cornea and crystalline lens are too much curved. Their refractive power is excessive, so that in an eyeball of normal length the light from distant objects is focussed in front of the retina.

In either case the refracting power of the eye is too great for its length. The common way to correct this is to neutralize a part of the refractive power by a concave lens. But from the earliest understanding of the subject, it must have occurred to any one who studied it carefully that myopia might also be corrected by taking away some of the refracting power of the eye. The crystalline lens is often removed for cataract; why should not the clear lens be removed to lessen the refraction of very myopic eyes?

A generation ago Mooren tried the removal of the lens for myopia, and reported favorable results at the Heidelberg Ophthalmological Congress in 1858. But no one supported his advocacy of the operation: Graefe strongly opposed it, and Donders condemned it. Mooren met with some unfavorable results, and gave it up. But in 1887 Fukala began removing the lens in young myopes where the defect was 15 D (dioptries) or over. His report of good results met with a favorable reception, and the operation was taken up and widely practised in Germany, one operator publishing one hundred and fourteen cases in a single paper. We must conclude that enthusiasm got the better of judgment in regard to the operation in Germany, for even the greater prevalence of myopia in that country could not explain the hundreds of cases that a few surgeons submitted to operation. In other countries the operation was accepted

with much more caution. In America only one case was reported (by Casey Wood, in 1890) prior to the report of my first case in February, 1898. Since that time several cases have been reported, and the question of the propriety of operation will now come up in every case of very high myopia.

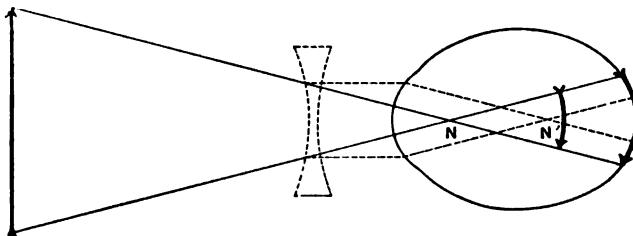
What does the operation promise in favorable cases? The myopia will be greatly diminished or changed to low hyperopia. The exact amount of the change that will be made in the refraction cannot be known before the operation. Usually it is about 17 to 18 D. It has been as little as 12 D and as much as 30 D in reported cases. It depends on the amount of the elongation of the eyeball and the part that curvature of the lens itself plays in causing myopia. Generally the higher the myopia the greater the change produced by removal of the lens. So if a myope of 15 D is made 2 or 3 D hyperopic by the operation, a myope of 25 D is likely to be left only 2 or 3 D myopic.

Such a diminution in the myopia enables the patient to escape from the annoyances of being dependent on very strong glasses; and these annoyances, as the myope knows, are real and great. They are so great that patients with very high myopia often refuse to endure them, and choose rather to suffer the practical blindness caused by their uncorrected myopia. Strong lenses are heavy. On account of aberration, they vary in effect as the eyes look through them obliquely. They greatly diminish the apparent size of objects seen through them. Their edges, acting like strong prisms, cause apparent displacement of objects, and produce fringes of prismatic colors about them.

The patient will not, however, be able after operation to dispense with glasses entirely. Very rarely will the operation correct the myopia so exactly that lenses cannot give marked improvement of vision. Even if distant vision could not be improved by any glass, after operation lenses would be required for near seeing, because the removal of the lens removes the power of accommodation, the power of focussing the eye for different distances. Then, too, the operation itself almost invariably leaves some corneal astigmatism, which must be corrected, to give the patient his best vision.

The acuteness of vision is generally increased, by removal of the crystalline lens, over that obtained by correcting the myopia with glasses, by reason of the increased size of the retinal images. How

these larger retinal images are gained may be understood from the following diagram which represents the formation of the retinal image in a myopic eye. At the nodal point, N , rays from different



points of the object cross and begin to diverge. The size of the image is proportioned to its distance from the nodal point. The shortest arrow in the figure represents the image which would be formed on the retina in an eye that was not myopic. In the emmetropic eye the retina is about fifteen millimetres from the nodal point. But in a case of myopia of about 20 D its distance from the nodal point is twenty-five millimetres, or one and two-thirds times as far. It receives an image that is two-thirds larger than the image formed in the emmetropic eye.

Now one effect of a concave lens placed before the eye is to carry the nodal point back to N^1 nearer the retina. The effect of a concave lens strong enough to correct the myopia when placed in the usual position in front of the eye, about one-half inch from the cornea, is to diminish the retinal image to the size of the retinal image in an emmetropic eye. This is illustrated by the broken lines in the diagram. This diminution in the retinal image is a necessary accompaniment of the correction of myopia by concave glasses. But when the myopia is corrected by removing the crystalline lens, there is no carrying back of the nodal point, no diminution in the retinal image. In the case supposed above, the image obtained by removal of the crystalline would be two-thirds larger than the image obtained with concave lenses, and the acuteness of vision would be that much greater after the operation.

As a matter of fact such an improvement of vision is often reported after operation. In one eye from which I removed the crystalline lens for myopia, the best corrected vision was improved from five-twenty-fifths to five-ninths, and in another from five-thirtieths to five-ninths. Now such improvement of vision is often of the highest

importance to the myope, whose visual acuteness is always lowered by the stretching, and the inflammatory and atrophic changes in the choroid and retina, that always accompany high myopia. Such an improvement may carry a patient from a condition where reading of ordinary print is almost impossible, to one in which the reading of ordinary print is easy. It may enable the patient to continue, without risk, in an occupation that, without it, would quickly have destroyed useful vision, by additional pathologic changes due to eye-strain.

It has been claimed for the operative treatment of high myopia that not only did the operation diminish the myopia, but that it also diminished the tendency to intraocular disease, always present in myopic eyes, diminished the tendency to further distention of the eyeball, and even brought about a progressive shortening of the eyeball so that the myopia continued to diminish for months and years after the operation. How far we are justified in expecting such benefits from this line of treatment is doubtful. It is very probable that the improvement in acuteness of vision, and the better conditions of eye-work brought about by escape from the need of bringing the eye very close to everything that has to be seen, or the removal of the difficulties attending the use of strong and generally imperfectly correcting lenses,—it is probable that release from these unfavorable conditions may cause marked improvement in the ocular health.

But with regard to progressive diminution of myopia subsequently, there is more reason for doubt, although many have claimed to have observed it. It must be remembered that after the extraction of the crystalline lens for cataract, the lack of resistance in the corneal scar lets the cornea bulge forward, thus elongating the eyeball as well as causing the recognized post-operative astigmatism. As the healing of the corneal scar becomes more complete, its resistance to the pressure of the fluid contents of the globe increases, and the cornea is retracted, with corresponding shortening of the eyeball and increase of hyperopia. This process may continue for many months. It occurs also after extraction of the clear lens for high myopia, and would fully account for the decrease of myopia that many observers have noticed in the first few months after operation. It is a process which is simply a part of the healing after operation, and not an ultimate influence of the operation on the nutrition of the eyeball.

Then, too, the observers who have reported on their cases several

years after operation do not find any continued progress in the direction of shortening of the eyeball. Indeed, in some cases, the further elongation of the eyeball has been unmistakable; the myopia, although greatly diminished, has continued progressive. It must be borne in mind, too, that after the removal of the crystalline a smaller increase in the myopia corresponds to the same lengthening of the eyeball. This is a practical advantage to the patient. But it may lead the surgeon to underrate the gravity of the progressive change in the eye, and overrate the influence of the operation. The hope that operative treatment would prevent detachment of the retina has also proven fallacious.

What eyes should be operated on? In the first place, only those that will not or cannot wear the proper correcting lenses. Some that have written upon the subject have made very light of the dangers of the operation. But I have seen two eyes lost from the operation performed by other surgeons, whom I know were careful and competent operators; and one of these eyes I had to enucleate after it had caused sympathetic ophthalmitis. Now, as I have only seen six cases in which the operation has been done, and as operators are slow to publish failures, except in connection with large series of successes which will somewhat balance them, it seems certain that the risks of the operation are important, and that it should not be resorted to if lenses can be worn with satisfaction.

The operation should be confined to eyes having over 12 D of myopia. If the myopia be much less than this, there is the chance that the patient may have to wear as strong a glass (convex) after the operation as he did (concave) before it. It is sometimes advised to operate on rather lower degrees of myopia if it seems rapidly progressive in young persons. But myopia even of high degree often ceases to be progressive under the constant wearing of correcting lenses. And even if it should continue to increase, the correction by operation is likely to be more exact and satisfactory after the myopia has exceeded 15 D than before. Perhaps the most satisfactory result is to leave the patient emmetropic, so that he will require glasses only for near work; or with a myopia of 3 D, so that near work can be done without glasses.

The operation should be confined to those cases in which decided improvement of vision is produced by correcting lenses. If the optical correction by lenses makes no material difference to the patient's

vision, little can be hoped from optical correction by removal of the crystalline. A good deal has been said about choroidal disease as a contraindication to the operation. When the fundus lesions are such that correction of the fault of focus produces but little improvement of vision, they are a decided contraindication; but short of this they do not prohibit operative treatment.

Some operators would confine removal of the clear lens to patients under forty. The greatest benefits can be conferred on young patients, who can quickly adapt themselves to the new conditions of seeing. Many of the patients who are sufficiently myopic to justify this operation, ere reaching that age will have suffered such intra-ocular changes as render operation useless; and in some the lens is no longer clear, so that its removal will really be a cataract extraction. But, if in all other respects the operative treatment seems indicated, I would not hesitate to adopt it on account of the patient's age.

In undertaking so important a measure, care should be taken to insure that the patient is in good condition for the operation. The general health should be at its best, and the eye should be free from active inflammation or evidences of very great degenerative changes. Thus, a fluid vitreous, or recent hemorrhage into the vitreous or fundus, should exclude the eye from operation.

Method of Operating.—No one method is suited to all cases. In early life repeated discission leading to absorption of the lens is to be preferred. This method entails no very extensive operation. The patient need not be confined to bed, and can use the other eye; and little or no post-operative astigmatism will result. The first discission should always be slight,—a single cut in the anterior capsule of the lens, not more than two or three millimetres long, with such stirring up of the lens substance as can be effected through such a cut without risk of enlarging it. Then the eye should be closed for a few hours, and kept continuously under the influence of a mydriatic.

After such a discission, the immediate disturbance caused by operation should subside in a day or two. Then if no secondary irritation or hyperæmia should arise from swelling of the lens, or from fragments of lens substance falling into the anterior chamber, the eye may be watched for some weeks until the lens-changes set up by the discission seem to have come to a standstill. By this time one can judge whether it is best to rely on repeated discissions. When the lens has become decidedly swollen and opaque, and the swelling has

been reduced, the next discussion may be much more free, extending five or six millimetres or more. After the bulk of lens has been absorbed, an incision should be made to extend through the posterior capsule.

If, after a discussion, considerable swelling of the lens is attended with hyperæmia and other signs of decided intraocular irritation, the bulk of the lens should be promptly extracted. In patients of twenty to twenty-five years this will be the method planned from the start. But in some patients of half the ages mentioned, if the whole lens seems to have become opaque, yet little be gained in the way of removal by absorption, the same procedure should be resorted to.

In removing the lens, we should seek to do it with the least mutilation, and so to place the corneal incision as to lessen or remove existing corneal astigmatism. If the lens has become thoroughly softened, a corneal incision six or eight millimetres long should be sufficient. It should be made with the lance-shaped keratome or a narrow cataract-knife, which may be used to make a free opening in the capsule. The wound may be held open with a curette or corneal spatula, and the lens substance pressed out. The object of the small corneal wound is to lessen astigmatism and the risk of prolapse of the iris. But the incision must be large enough to allow the free escape of the lens substance. Even when it is intended to extract the lens so soon as it has become opaque, the first opening in the capsule should be small. Of the cases I have seen, all that did badly had the lens freely divided at the first operation, as recommended by Fukala. This was followed very soon by pain, redness, and other symptoms of inflammatory reaction. Even in cases thus treated which ultimately gave favorable results, the tendency to inflammation was manifest throughout the course of the case, and the eye became entirely quiet only after many weeks. On the other hand, while the small incision in the capsule may seem at first to produce little effect, the opacity and alteration of the lens is more complete, because one is not forced by the inflammatory symptoms to give the eye early relief from the effects of the excessive swelling of the lens; and the healing after removal of the lens is more prompt and satisfactory.

After the age of forty or fifty, it is probably best to make the primary operation an extraction of the clear lens. Such an extraction will be more or less incomplete; but it is not difficult to remove the nucleus and a good deal of the cortex. In the main, the tech-

nique of an ordinary cataract extraction is to be observed; with especial care to make simply a linear opening in the lens capsule, parallel and as close as possible to the corneal incision. This will insure the smallest escape of lens substance into the anterior chamber; and after the removal of the nucleus the changes in the clear cortex remaining in the capsule will do little harm. Should a considerable amount of cortex get into the anterior chamber, it may be worth while to wash it out with Lippincott's or some other form of intraocular syringe.

Of course, by this plan, there will remain enough cortex to block the pupil and prevent vision until, by a second operation, a clear opening is made through the remains of the lens and its capsule. But by getting rid of the nucleus at the first operation the most serious risks of removal of the clear lens in elderly people are avoided. The same plan may be followed in younger patients, but does not offer for them the same special advantages.

Should both eyes be operated on? Where the case is one especially demanding operative treatment, where the uncorrected myopia renders the eye practically useless, and both eyes present the proper indications for operation, it may be proper to give the patient the benefit of improved vision in both. But only one should be operated on at a time. The eye first attacked should have some months to recover fully from the operation, and show just what had been accomplished with it, before anything is done to the other. To deprive the patient of the use of either eye for several weeks, and to risk both by simultaneous operations, is quite unjustifiable. Generally the worst eye should be operated on first, although it must still be good enough to be capable of decided benefit by such treatment.

In some cases one eye is practically lost by the time the myopia becomes high enough to be properly subjected to operative treatment. These cases are not very favorable for operation. Still, in the absence of evidence of especial danger, it is sometimes proper to operate on them. Sometimes, as in cataract cases, after one successful operation the patient desires that the other eye shall be let alone. In that case he cannot be strongly urged to a second operation. The eyeballs, although freed from myopia, remain elongated, and poorly suited to binocular vision, and are in danger of farther progressive distention if subjected to the pressure exerted by the extraocular muscles in convergence.



FIG. 1.—CASE I. Four nails affected by favus.



FIG. 2.—CASE I. Cured.

THE TREATMENT OF FAVUS OF THE NAIL.

BY FRED. J. LEVISEUR, M.D.,

New York.

DR. LEOPOLD GLUECK, of Sarajevo, Bosnia, reports¹ to have observed five cases of favus of the nail and three of favus of the body among a total of two hundred cases treated in a period of four years. This report, coming from a country where favus is a common disease, shows how rarely the fungus attacks the nails. In this country, where favus is certainly not very prevalent, onychomycosis favosa is such a rarity that descriptions of this affection, as found in our text-books, are mostly copied from continental writers; and as regards treatment very little of all that is recommended is based on actual experience. I had occasion to observe three undoubted cases in which I was able to make the diagnosis clinically as well as microscopically. In every case the nails were attacked secondarily, the scalp showing either fresh symptoms of the disease or its disfiguring results: irregular, bald, slightly depressed, and glossy spots.

The surface of the nails was affected comparatively little, and seemed to be only mechanically injured and not affected directly by the disease. In this regard the affection differs materially from eczema, psoriasis, syphilis, and perhaps ringworm of the nail. There was always evidence that the disease had started from underneath the free edge of the nail, and, thriving in the rete mucosum, had lifted the horny plate of the nail upward.

It has long been recognized that the mechanical pressure of the fungous masses in their process of development is extraordinarily strong. In mice, which are particularly prone to acquire favus, scutula, or favus-cups, sometimes produce perforation of the skull and consequently prolapsus cerebri. It is, therefore, not surprising

¹ "Ein Fall von Favus am Penis," Archiv für Dermatologie und Syphilis, xlvi. 8.

that in favus of the nail the fungous material enters like a wedge between the horny and the papillary layer. It attacks at the same time principally the deepest and softest stratum of the horny nail. In comparing *a* with *b* (Fig. 3) it will be seen at a glance that it is the inner surface of the nail which is attacked by the disease in this manner. After this fact had once been recognized no doubt remained as to the most effective treatment. It consists chiefly in the removal of the affected nail. The avulsion of the nail would be a severe procedure, if attempted in an early stage of the disease; but it becomes very easy and almost painless when the affection is allowed to spread until it has reached the lunula, the patient during this time taking care not to injure the horny shell which is thus formed. If this is accomplished the nail can easily be pulled out of the epidermal fold. Freezing the finger with methyl-chloride spray will render this little operation absolutely painless. It will be followed by very little, if any, bleeding, which will promptly stop under the application of a cotton collodion dressing. The application of an antiparasitic salve, for instance, ten per cent. of salicylic acid, is all that is necessary after the removal of the nail. The new nail will soon make its appearance, and, by pushing forward, smooth over, as it were, the rough and ridgy surface of the bed of the nail. Four fingers of Case I. (Fig. 1) were treated in this manner, with the result shown in Figure 2. A great many remedies had previously been tried without effect for a period of seven months. A second case (Fig. 4) was treated at once in this manner. The patient, a boy of twelve years, was advised to keep the hollow space underneath the nail clean and prevent the breaking of the horny shell. After five weeks the latter could be removed with the greatest ease, and the new and healthy nail soon appeared (Fig. 5). In conclusion, I want to say that, though it may be possible to stop the progress of the disease at a very early stage by other means, the best treatment for somewhat advanced cases is to permit the disease to lift the nail from its bed until it can be, and then is, easily removed. It is hardly necessary to say that the other nails should be carefully protected against infection, and that just for this reason thorough treatment of a co-existing favus of the scalp is of the greatest importance.



FIG. 8.—Nails after removal; *a*, outside.



FIG. 8.—*b*, inside.



FIG. 4.—**CASE II.** Nail of little finger undermined by the disease, the surface having retained its normal gloss. Bald spots of the scalp the result of favus.

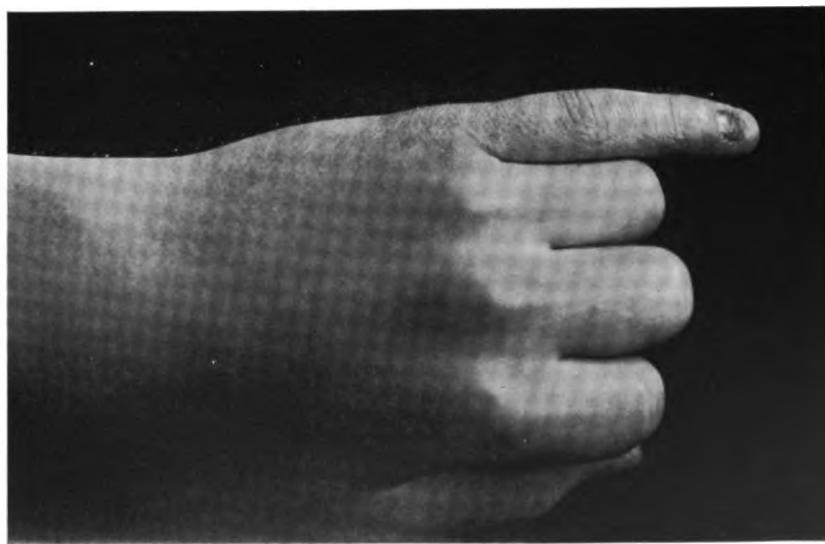


FIG. 5.—**CASE II.** Little finger of the same patient four weeks after removal of the nail, showing the growth of new and healthy nail.

Medicine.

DISTURBED CIRCULATION AND ITS EFFECTS ON THE BRAIN.

CLINICAL LECTURE DELIVERED AT THE LONDON HOSPITAL.

BY FRANCIS WARNER, M.D. (Lond.), F.R.C.P., F.R.C.S. (Eng.),
Physician and Lecturer on Clinical Medicine and on Therapeutics in the London
Hospital, England.

GENTLEMEN,—Healthy organs in the body and a brain that has previously performed healthy functions may become greatly disturbed by temporary disorder of the circulation. I wish to draw your attention especially to the mental, sensory, and motor disturbances that may follow from altered conditions of the circulation in the brain as the main cause. It is a great mistake to give an opinion that the brain itself is defective in its tissues or structure or undergoing some form of degeneration when the disorderly action in it is due mainly or solely to vascular changes which may be of temporary duration or easily avoidable.¹ In other words, we should not mistake a healthy brain with a disturbed circulation for a defective or degenerate organ unless we find direct signs that the brain itself is the seat of disease or congenital defect.² I shall not attempt to deal fully with the physiology of the intracranial circulation, but will draw your attention to a few facts and to some clinical points commonly met with where the conditions are obvious enough for study and inference.

¹ See author's article on "Neural and Mental Disorder in Children," Cyclopaedia of the Diseases of Children, Supplement, J. B. Lippincott Company.

² See "The Signs to Observe in Brain Disorder in Children," INTERNATIONAL CLINICS, 1896, vol. ii.

The vascularity of the brain tissue may vary. This is well shown in the following photographs, taken from the beautiful colored drawings published by Sir George Burrows in his volume "On Disorders of the Cerebral Circulation," 1846, from which I have taken two examples (Figs. 1 and 2). "These drawings represent the upper surfaces of the brains of two rabbits. The one animal (*A*) was destroyed by hemorrhage; the other (*B*) by strangulation. The contrast between the two heads in point of vascularity, both on the exterior and the interior, is most striking. In the one (*A*) scarcely a trace of a blood-vessel is to be seen; in the other (*B*) every vessel is turgid with blood."

A normal blood-pressure in the arteries and uniformity in the capillary circulation, as well as the quality of the blood, are important elements to the healthiness of the brain. The variations in the blood-pressure of the brain, and its effects, may to some extent be studied conveniently in the clinical observation of infants—say, about nine months old—in whom the head has attained a fair development, while the fontanelle is still patent; such an infant should present a cranial circumference of about seventeen and a half inches. At this age the great characteristic of healthy brain action is abundant spontaneity, together with some co-ordination through the senses, as shown in prehensile action and the conveyance of objects from hand to hand or to the mouth.¹ This is found only in healthy infants with a sufficiently good brain circulation, as indicated by pulsations of the fontanelle, which also by its moderate convexity shows the general blood-pressure in the head. Movements of the fontanelle are affected by the respiration as well as by the beats of the heart; venous pressure increases with expiration, as is seen in the act of crying, which markedly raises the fontanelle, while the face becomes flushed.

When an infant is ill, as from diarrhoea or broncho-pneumonia, the blood-pressure falls and causes a marked degree of brain shrinkage, indicated by depression of the fontanelle and loss of its pulsation. This is accompanied by loss of spontaneous movements. The sick child is nearly motionless; he cries, but does not grasp an object; the eyes move, but there is no prehensile act; failure of the brain circulation results in diminution of brain function. On recovery of a

¹ See author's "The Study of Children and their School Training," p. 48, The Macmillan Company.

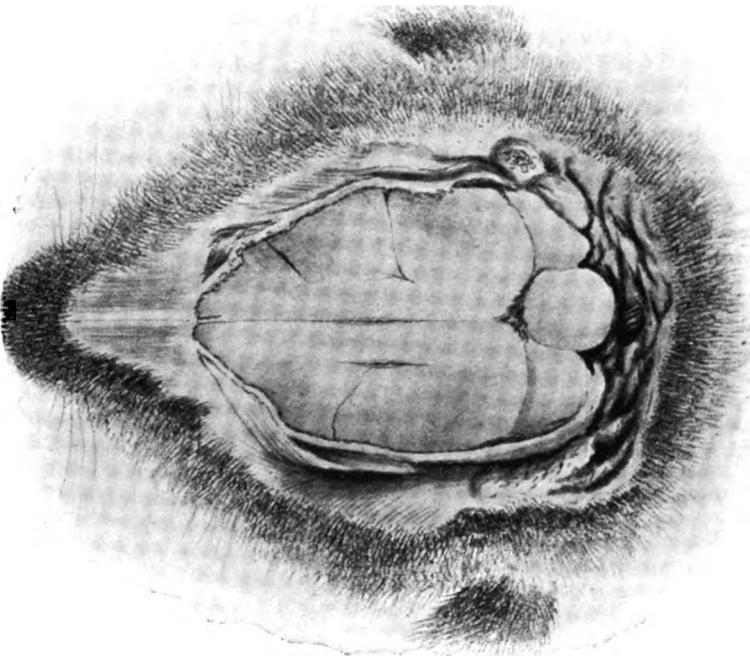


FIG. 1.—Hemorrhage.

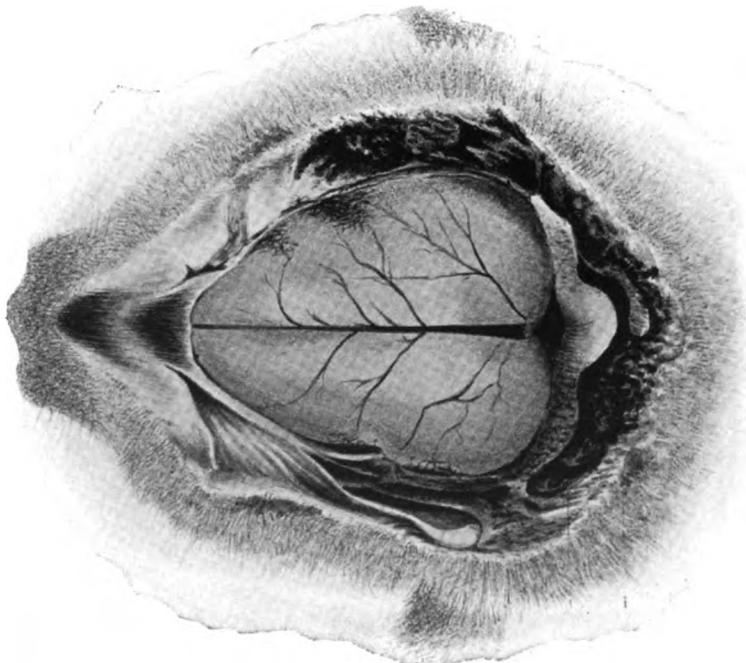


FIG. 2.—Strangulation.

healthy brain circulation, spontaneity is restored, as seen in the lively movements of the face and limbs.

I will now narrate a case in which grave mental inaptitude occurred in a school-boy, and lasted about twelve months, apparently dependent upon functional disturbance of the circulation, which affected both the brain and the kidneys, while there was no real defect or disease in those organs.

CASE I.—A boy, ten years of age, was receiving his education at a private boarding-school, where he had been for half a year, and was looked upon as a quick and promising pupil, being nearly the youngest among fifty others. He was accustomed to play cricket, hockey, and foot-ball, and he went to the swimming-bath and had drilling-lessons. During his second term of attendance at school he showed serious signs of failure in memory with mental inaptitude. He could not repeat in class the lessons learned overnight, and forgot what he had been told to do, and really did no work; as the holidays were near at hand, this was passed over. His mental failure was believed to be the result of his coming into collision with another boy while running to the play-ground; he fell, but there was no loss of consciousness; the evidence as to this accident was uncertain. In the vacation he was languid and was not allowed to work. During the next school term his power of memory was very defective and at times he seemed not to know what he did; but, though forgetful, he never did anything wrong, and no kind of attacks were observed. Six months from the commencement of this failure of power his memory and application to study remained very weak; the urine was now examined, and found to contain a trace of albumen. He was removed from school; massage was employed at home, and in three weeks he dropped three pounds in weight.

Shortly after this I first saw the boy. On inquiry the history as given above was elicited. The lad had always been healthy, though rather thin. He had two sisters and one brother, all older than himself and well in health.

He was rather tall, but slight in figure; height four feet and ten and a half inches; weight sixty-nine pounds (normal at ten years: height four feet and five inches; weight sixty-two pounds.—BOWDITCH). His head was well shaped, circumference twenty and three-quarters inches, transverse measurement fourteen inches, from occipital protuberance to bridge of nose thirteen and a half inches. The

palate was well formed, and the teeth were not ground. The features were well moulded, and the skin and mucous membranes were healthy. His speech, balance of body, and response in movements or in imitation of my action were good. His expression was bright, and he had not the fulness under the eyes which often expresses fatigue. There was some blinking of the eyelids, unaccompanied by conjunctivitis. The reaction of the pupils was normal; the reflexes were natural, and no abnormal nerve-signs were present as indicative of brain deficiency or disease. In mental examination he gave clear answers to questions and a good account of his school life and holidays; simple calculations he made correctly. In short, there were the signs of good brain action well trained. On further examination, expansion of the chest was rather weak, with a small respiratory capacity, but the lungs were healthy. The heart-sounds were clear, but the impulse was strong and the pulse quick, as from nervous excitement. His lips were only partially closed; though no naso-pharyngeal obstruction was detected, it may be that attention to this will be required; it appeared unadvisable at the time to make a complete examination, as the boy was rather timid.

The urine which was brought with him had been passed on rising in the morning, and was quite normal, but that passed at the time of the visit contained a trace of albumen. Numerous further samples were forwarded to me, but none contained any albumen; sometimes the urine was clear, but often there was a deposit of lithates without any crystals; the average specific gravity was 1020. In fact, the kidneys appeared to be healthy. On the other hand, the water passed at each visit to my house contained a trace of albumen, and the same fact was noticed on his visit to another physician.

During the next three months he was kept from school and did no work. At times he was emotional, thinking his friends unkind, and would burst into tears; he was sometimes excited, especially if fatigued. He preferred a bicycle to walking, as less fatiguing. Sleep was not bad, but he moved much in bed and sometimes called out; he would talk about his dreams without being questioned. The appetite and digestion were healthy.

The weight had now fallen to sixty-seven pounds, but his general health was good. On advice he returned to his school ten months after the commencement of the symptoms. At the end of this term, one evening during the examinations, he was unwell, and his temper-

ature was found to be 101.6° F., rising the next day to 104°, and falling on the third day to 100°, when I saw him, and found the throat and lungs clear, though the urine again contained a trace of albumen. There was not sufficient evidence to account for this temperature other than disturbance of his nervous system. A year previous he had a similar attack of high temperature, after a long journey, without any other cause discoverable. His weight was now sixty-six and a half pounds.

At the end of fifteen months from the onset of the mental symptoms he was reported as being in good health and again working satisfactorily in school. His weight had risen to seventy-one pounds.

The clinical aspects of this case are interesting. They present a boy well made, but slightly delicate, with occasional temporary albuminuria but healthy kidneys. There were no signs of brain defect and no real lapses of mental action, but failure of memory with mental confusion. I may here add that careful means were taken by a skilled observer to ascertain that there were no attacks of *petit mal*. The patient was of the nervous or brain-mobile type, always liable to paresis of the centres for cardiac inhibition, leading to a rapid pulse, which sometimes reached 120 per minute, while this apparently caused such disturbance of the brain circulation among the higher centres as to interfere with their mental functions, as well as with those of the kidneys. Another pair of brain-centres liable to disturbance appear to be those which are situated in the neighborhood of the corpora striata and control temperature in the body. The case appears to be one of primary disturbance of certain brain-centres which, affecting the circulation, lessened the functions of the cortex cerebri. There was no general motor disturbance, such as occurs in the various forms of spreading action in the brain, following sensory stimulation and producing many extra movements, a condition often called "nervousness."

A favorable prognosis was given in this case, which thus far has been confirmed.

The brain tissue in its cells and fibres needs an average amount of arterial blood-pressure and an equitable circulation in its capillaries, the blood itself being of such quality as is suited to normal nutrition. In any of these particulars a disturbed blood-supply may lead to brain symptoms. In a former clinical lecture I narrated cases where valvular disease of the heart produced cerebral disturbance by

affecting the capillary flow; other mechanical conditions may produce the same effect.

Any cause impeding the outflow of venous blood from the brain tends to arrest its capillary flow and interferes with its functions. You have often seen cases of chronic bronchitis in emphysematous patients with dilatation of the right heart, in whom during a prolonged attack of coughing the venous obstruction is very great, the jugulars standing out prominently in the neck, while the face is purple, at which stage the patient may become temporarily unconscious and fall against the wall before he recovers himself. Attacks of whooping-cough may lead to convulsions, and occasionally to hemorrhage on the brain.

CASE II.—You recently saw in the wards a man with mitral disease and great congestion resulting; the liver was large and accompanied by considerable ascites, while the legs were œdematosus; the urine was scanty and contained albumen. The patient complained much of head pain with sleeplessness, and had an anxious expression. The veins in the neck were prominent, and I directed venesection of the right external jugular, which after a flow of sixteen ounces relieved his head distress so greatly that he slept well, though the previous employment of hypnotics had failed to give him rest.

CASE III.—Some of you may remember a man in the hospital last year with extreme cyanosis and œdematosus legs that had sloughed extensively, who became completely comatose from heart failure, yet recovered consciousness after fifteen ounces had been drawn from his jugular.¹

Pathological conditions of the blood that circulates may produce marked disturbance in a brain previously healthy. In uræmia the blood-pressure is often high, and the blood itself impure; headache, vomiting, twitchings, convulsions, and coma may supervene.

CASE IV.—A man was admitted here with severe headache accompanied by the aspect of suffering and vomiting. There were the usual signs of hypertrophy of the left ventricle of his heart; the tension of the radial pulse was markedly above the normal; the urine had a specific gravity of 1010 and contained a trace of albumen. As there was no history of previous head-attacks in this case and no abnormal nerve-signs were observed beyond the facial expression, it

¹ Case given in INTERNATIONAL CLINICS, 1898, vol. iv., "Venesection in Heart Failure."

appeared probable that uræmia, or an impure condition of the blood, was the cause of his head distress. The hot-air bath (120° F.) failed to produce sweating, but when this was supplemented by the hypodermic injection of one-sixth of a grain of nitrate of pilocarpine free diaphoresis followed within two minutes. This treatment was repeated; his head pain was entirely relieved, and the blood-pressure fell to a normal standard and remained so while the man was under observation.

In enteric fever the blood-pressure falls while the temperature may be high; the brain, previously healthy, becomes markedly disturbed, the patient falling into a somnolent condition, with loss of spontaneity and making but little response, while delirium and finger-twitches or subsultus tendinum may supervene.

Sensory impressions and sometimes sudden alteration of atmospheric pressure may suddenly accelerate the pulse and so far interfere with a normal circulation in the brain as to produce serious disturbance. In some patients of the nervous type, at the commencement of an interview, the pulse rate will be found to be 110 per minute or higher, and their talk may show some mental excitement or confusion, without any extra movements or spreading area of motor action such as indicate general (motor) excitement; this was the case with the school-boy already described. I usually count the pulse at the commencement of an interview and again later on.

CASE V.—A lady, thirty-four years of age, consulted me on account of an attack of syncope which occurred under the following circumstances. Travelling with her family to Switzerland on a pleasure trip, she stayed at a hotel about six thousand feet above sea-level. Five days after arriving there, and without having gone through any unusual fatigue, she became suddenly very faint, was blue about the mouth and quite powerless, but consciousness was not completely lost, while there was a sensation as of roaring in the ears. No indigestion or over-exertion accounted for the attack, and none such had previously occurred. On examination, I found health and digestion in good order; there was no anæmia; this lady was accustomed to walking and cycling in the country without any difficulty; she was not a neurotic patient and did not suffer from headaches. The pulse was quiet and regular; there was a systolic apical murmur over the heart, evidently due to slight mitral regurgitation, but without indications of ventricular dilatation. The sudden attack of syncope appeared

due to the effects of lessened atmospheric pressure, disturbing the heart and producing anaemia of the brain. I saw this lady a year later for a trifling ailment; no further syncopal attacks or heart disturbance had occurred.

I have shown that a disturbed circulation may render the brain disorderly in action; I must remind you that defects in the circulation may be secondary to nerve-disease, and in illustration will only mention the rapid circulation of Graves's disease, the slow and intermittent pulse of early tubercular basic meningitis, and the low-tensioned pulse of small volume in brain exhaustion, as after an attack of acute mania or an epileptic fit. This shows the necessity, in a patient with nerve-disturbance and disordered circulation, of examining the signs of each system separately and comparing them before forming an opinion.¹

In functional cases it is not always easy to differentiate between primary brain disturbance and the effects of disordered circulation; indeed almost always they interact on one another. Cases of migraine are common; vascular disturbance often accompanies the attacks. A pair of arteries, as the temporals or occipitals, may be tetanized, and can be felt by the observer as hard and cord-like, attended with great pain. On the other hand, the face may be flushed and one ear red, from a neuro-paretic condition; or the features may look pinched and shrunken. Any movement of the head increases the pain, while coughing produces acute suffering, as the blood-pressure in the veins is increased. Hence during the attack you keep the patient in the horizontal position, to equalize the circulation, and apply warmth to the feet.

In the hygienic control of healthy brains due means are required to keep up a healthy standard of the blood and also a good circulation. This is specially important with children during their school-life, and not less so with men and women students in their college career. Physical exercise promotes brain nutrition in the healthy; delicate children sometimes work best when sitting in a chair which gives some support. The man with a tired brain quickens his circulation by pacing the room as he thinks. Dr. Lauder Brunton,² referring to the soothing effects of tobacco on the nervous system and

¹ See INTERNATIONAL CLINICS, 1898, vol. iii, "Physical Signs in Examination of Brain Cases."

² Pharmacology, Therapeutics, and Materia Medica, 1887, p. 998.

its stimulating action for mental work, attributes the result not to the nicotine itself, but to the stimulus of the smoke on the sensory nerves of the mouth, which reflexly stimulate the vasomotor centre and dilate the vessels of the brain. To some extent sipping warm drinks and tea produces a similar stimulation, effected by reflex action causing dilatation of the arteries supplying the brain.

I trust that what has been said has shown distinctly that, in making a diagnosis in the case of a patient complaining of nerve-symptoms, you should look carefully to the nerve-signs indicating the brain condition,¹ and also to the state of the circulation, disturbance of which may cause great disorder in a brain otherwise healthy.

¹ See author's article on "Scientific Study of the Mental and Physical Conditions of Childhood," Cyclopaedia of the Diseases of Children, Supplement, J. B. Lippincott Company.

SYPHILIS OF THE KIDNEYS; SYPHILITIC NEPHRITIS; FORMS AND TREATMENT OF SYPHILIS OF THE KIDNEYS.

CLINICAL LECTURE DELIVERED AT THE HÔTEL-DIEU, PARIS.

BY PROFESSOR DIEULAFOY,

Professor of Internal Pathology, Faculty of Medicine, Paris,

AND

BY A. F. PLICQUE, M.D.,

Chief of Laboratory, Lariboisière Hospital, Paris.

SYPHILITIC NEPHRITIS.

GENTLEMEN,—Before presenting to you the patient who is to form the subject of my lecture to-day,—a young man with a serious case of syphilitic nephritis,—I must first say a few words as to whether this morbid entity exists in reality.

In 1885 a German writer, Guns, endeavored to show that syphilis could not give rise to nephritis, but that the cases reported as such were nothing but kidney disorder caused by mercury; basing his opinion on experimentation on animals, he claimed that mercury produced amyloid degeneration of the kidneys, which was the point of departure of the cases of nephritis observed in syphilitic patients.

This opinion made a great impression at the time, and caused a number of physicians to abandon the use of mercury in syphilis; since then, however, a reaction has taken place against Guns's opinion, which, if it was not absolutely erroneous, was in conflict with a large number of facts.

Thus, a man with sound kidneys develops a hard chancre, and six or eight weeks later is stricken with albuminuria and anasarca without ever having taken mercury. Another case will show no signs of nephritis until the tenth or twelfth year of his syphilis; he was treated

with mercury, but not later than the fourth year of the disease, and without having shown at that time any signs of nephritis. It is not possible to blame the mercurial treatment for nephritic disorder as tardy as that. Finally, we frequently see in our wards workmen suffering from professional mercurial intoxication, many of them deeply impregnated with this poison, and yet they are not albuminuric. If Guns's opinion were true, there ought to be a mercurial nephritis just as there is a saturnine nephritis.

Consequently, we will set aside Guns's assertion, and say merely that when nephritis already exists when mercury has to be given, the elimination of this remedy may be hindered, and mercurial intoxication ensue as a result of the nephritis, which is therefore a cause and not an effect.

Another point that must be carefully borne in mind is that syphilitic nephritis, usually an early complication of the disorder, is a diffused, epithelial nephritis, and, as such, must be clearly separated from the tardy kidney complications of syphilis, which belong to the types of gumma or sclerosis, or to a combination of the two. These tardy forms may affect one kidney only, or even only a part of one kidney, whereas the early form of nephritis affects the whole of both kidneys.

The importance of this distinction is connected mainly with the prognosis, which, in early epithelial nephritis is infinitely more serious than in the tardy form with gummatous and sclerosis. The prognosis of the early form is a terrible one, like that of all acute epithelial inflammations of the kidney, such as scarlatinous nephritis, than which it is even more sombre.

Our patient to-day is a young man of seventeen, who had not long ago an ordinary hard chancre of the penis, of which a slight induration can still be felt, and an unimportant roseola of the skin; later on came the usual ganglia and mucous patches of the mouth and anus. The case was a manifest one, and it is now exactly at the sixty-sixth day since the chancre appeared. When he entered our wards he showed enormous oedema of the legs, reaching up to the lumbar region, and his urine contained the extraordinary amount of twenty-three grammes, or six drachms, of albumen per litre or quart.

We have, then, here an early case of nephritis occurring in the third month after syphilitic infection in a young man who has never

been treated and who has taken no mercury. This case, you will see, is in absolute opposition to Guns's ideas.

I put him on a mixed treatment, biniodide of mercury and iodide of potassium, with a milk diet, and the chart which you see shows that day by day there has been a parallel decrease in the patient's weight and the quantity of albumen in the urine. This does not indicate that our patient has really lost weight; it merely shows that the œdema has decreased and the liquid stored up in the subcutaneous tissues has been eliminated. His weight went down from sixty-six to fifty-eight kilos, or from one hundred and thirty-two pounds to one hundred and sixteen pounds, and the albumen from twenty-three to two grammes, or from six drachms to thirty grains. Such has been the effect of this treatment, and we may look forward to the œdema soon disappearing altogether, and the albumen also. This is always the course of this disorder when it goes in the right direction.

This patient showed neither headache nor disorders of the sight or hearing; the disorder revealed itself solely through anasarca and albuminuria. It yielded readily to treatment, and did not assume the very serious aspect of many cases of syphilitic nephritis, for you must not imagine that all of these cases end so satisfactorily.

There are certain cases where you will find no more than headache, lassitude, coldness of the extremities, and disordered hearing, all of which the physician may lay to the score of the syphilis unless analysis of the urine reveals their true origin. You must be prepared for cases of this sort, where there may be no anasarca, or, at any rate, nothing but very slight œdema of the feet and eyelids.

On the other hand, there are many acute and very serious cases, and a certain number of these patients die from syphilitic nephritis in the fifth or sixth month of the infection. It is not always possible to cure these attacks, and at first the prognosis of our patient was distinctly serious. Many of you will remember that man of thirty-two who occupied, a few months ago, a bed very near to that of our present patient, and who entered our wards with nephritis in the sixth month of his syphilitic infection, with anasarca and masses of albumen.

I may say in parenthesis that this enormous anasarca and great quantity of albumen are characteristic of syphilitic nephritis, and

from this point of view no other form of nephritis save that of scarlatina can be compared with it.

When our former patient, the man of thirty-two, entered our wards, he weighed seventy-four kilos, or one hundred and forty-eight pounds, and showed sixteen grammes, or four drachms, of albumen. He was treated with injections of biniodide oil, though without much hope of saving him; his weight increased to seventy-five, seventy-nine, and eighty kilos, or one hundred and fifty, one hundred and fifty-eight, and one hundred and sixty pounds, and the albumen rose to nineteen, twenty, and twenty-three grammes, or four and three-quarters, five, and six drachms. After a few days the treatment was changed to potassium iodide, but without result; the weight rose to eighty-two kilos, or one hundred and sixty-four pounds; and although the albumen lessened a little, descending to twenty and eighteen grammes, or five and four and a half drachms, there was no real improvement, the œdema increased, respiration became labored, a lymphangitic flush appeared all over the chest and abdomen, dyspnœa set in, and the patient died a few hours later. Section showed the large, white kidneys of diffuse, parenchymatous nephritis, and in the right side of the heart an enormous clot, which must have been there for thirty-six or forty-eight hours before death.

Another young man whom I took care of a few years ago with one of my colleagues was suddenly stricken in the sixth month of a mild case of syphilis, with nephritis indicated by nausea, vomiting, general anasarca, and thirty-five grammes, or eight and three-quarters drachms, of albumen; the œdema was so intense that the tissues seemed on the point of splitting. This patient had taken no mercury, nor did we give him any at first; potassium iodide was administered, but in a few days the pulmonary œdema and dyspnœa increased. Mercury was then added to the potassium iodide, and slight improvement occurred, but it was soon followed by ascites and bilateral effusion in the pleura. Although this patient was threatened with pulmonary œdema, we ventured to tap his chest, only withdrawing about seven ounces of liquid. He felt better after this intervention, and several other punctures were made in succession, withdrawing from eight to fifteen ounces, each of which seemed to produce considerable relief. In spite of everything, however, after three months of nephritis (nine months of syphilitic infection) the patient died from ureæmic coma.

Here you have two instances of the serious form of early syphilitic nephritis, occurring during the first few months of infection. All of the cases of this nature that have been published are just alike.

The following is a case seen by Perroud, of Lyons (1882).

A man developed a hard chancre, followed by roseola in two months, ganglia, and then generalized papulous syphilis; he had taken no mercury, and was suddenly seized with general anasarca and showed large quantities of albumen. He was then treated with biniodide, but erysipelatous lymphangitis set in, with intense dyspnoea, and he died five days after this final manifestation.

Two cases were published by Wickham (1886).

1. A man of forty-two. Three months ago had hard chancre; the last six weeks, papulo-squamous syphilis all over the skin. In the second month of his infection, generalized œdema, affecting even the abdominal wall, and rising to the axillæ; effusion on both sides in the pleural cavities; six grammes, or one and a half drachms, of albumen. Had never taken any mercury. Two pills a day of half a grain of protiodide were prescribed, and from that time on steady improvement set in, ending in recovery.

2. A woman of twenty-one. Had hard chancre eighteen months ago, followed not long after by secondary symptoms. Has now twelve grammes, or three drachms, of albumen, and is put on the specific treatment for the first time. A month later, left the hospital, having only one and a half grammes, or twenty-three grains, of albumen; has since been seen again, quite well.

Another case, young man of twenty-one. Had hard chancre six months ago. At present, papulous syphilis; general œdema; liquid around both lungs; twenty grammes, or five drachms, of albumen (has never taken any mercury); erythema of the hips and scrotum. Recovered.

Other cases are to be found in Mauriac's very remarkable memoir on renal syphilis, published in 1887. Nephritis occurring (a) three months after the chancre,—death; (b) in the eighth month; (c) on the fifty-fifth day; (d) in the fifth month, that nearly ended fatally and lasted four and a half years; (e) two and a half months after the chancre.

Backer has published a very interesting case,—nephritis occurring in the sixth week after the appearance of the chancre in a young man of nineteen. Mercurial treatment; recovery.

Jaccoud published in 1887 a case of syphilitic nephritis that occurred at a very early date and lasted four years.

We have, therefore, sufficient data at our disposal to know what the characters are of this syphilitic nephritis: it appears suddenly, in mild cases of syphilis, in people who have had no renal disease and who have taken no mercury, and its chief characteristic is its early date of appearance.

Thus, if we take the dates at which it appeared in the cases published, we find, in the eighth month, two; in the sixth, two; in the fifth, one; in the fourth, two; in the third, three (including our patient); in the second, five. It is well to bear this fact in mind, as when a patient has reached the end of his first year of syphilis, this nephritis becomes exceptional.

Another peculiarity of syphilitic nephritis is the phenomenal amount of albumen contained in the urine of these patients. Our patient, twenty-three grammes, or six drachms; our former patient, sixteen grammes, or four drachms; the patient I took care of with my colleague, thirty-five grammes, or eight and three-quarters drachms; six, twelve, and twenty grammes, or one and a half, three, and five drachms, in Wickham's cases; but the record is held by a patient of Chantemesse, who showed fifty-three grammes, or thirteen drachms, followed by death.

The cases we have gone over show that, in addition to their early appearance and large amount of albumen, this form of nephritis is distinguished by its sudden appearance, the enormous oedema, the frequency of pleural effusion, as well as in the other serous cavities, the patient being little better than a sponge; finally, by their rapid evolution. In the fatal cases the patient died in two and a half months, or in five, six, or seven weeks, and death does not occur from uræmia. The patients die saturated with serum, through the abundance of the effusions, or by pulmonary oedema; often by secondary infection, such as erysipelas.

As regards the pathological anatomy of the disorder, this form is a diffuse, parenchymatous nephritis, the tubuli contorti are the parts more affected; and Henle's epithelium undergoes fatty, granular degeneration. There is usually no glomerulitis. To sum up, then, remember that:

1. Syphilitic nephritis is an *early* accident.
2. It is always serious, no matter how mild the syphilis.

3. Its two great characters are the enormous œdema and the large quantity of albumen.
4. It is always a diffuse, epithelial nephritis.

FORMS AND TREATMENT OF SYPHILIS OF THE KIDNEYS.

Syphilitic infection gives rise to an entire series of very different lesions of the kidneys. During the secondary stage it produces a sub-acute (or even acute), generalized infection, almost similar to that of scarlet fever. At the tertiary stage it creates more circumscribed and less dangerous lesions, either really specific, such as the gumma, or of a more ordinary nature, such as interstitial or parenchymatous nephritis, or waxy degeneration. Hereditary syphilis, both the early and tardy forms, can also affect the kidney, and paroxysmic haemoglobinuria is connected with syphilis in about one case out of two.

From this will be seen that the question is a complicated one; but happily the often extraordinarily efficacious action of the treatment, establishes a connecting link between these so very dissimilar manifestations.

Syphilis of the kidneys is often an extremely early localization (in one case it was observed at the fifty-third day of infection), and is very insidious. Pain in the lumbar region is hardly to be detected; at the beginning it is rather the disseminated œdema, or in some cases the haematuria, that attracts attention. The quantity of albumen is very large, and has been known to reach thirty-six grammes, or nine drachms, *per diem*. If the cause of this nephritis is not understood, the case may be extremely serious and the patient die in a few weeks from general anasarca or ureæmic complications. The haematuria is in some instances sufficiently abundant to cause death by itself.

The specific treatment, on the other hand, will cure it in nearly all cases, and this recovery may be very rapid, in a few weeks or months. Mauriac claimed that this rapid action of the treatment is one of the chief elements in the diagnosis. Still, in some instances the treatment has to be continued for several years. Transformation into chronic nephritis or waxy degeneration is rare. As is the case after all serious attacks of nephritis, great precautions are necessary to prevent recurrence; two things particularly to be avoided are blisters and exposure to cold.

Rayer's advice, therefore, to look for syphilis systematically in

all cases of nephritis is of great practical importance. Eruptions on the skin or mucous membranes, which often accompany the albuminuria, will greatly help in the diagnosis. Other causal factors may accompany the syphilis; thus, the patient may claim to have been exposed to cold, though the chill mentioned may merely have been the first manifestation of the syphilitic nephritis.

The part that was for a time supposed to be taken in the production of this nephritis by the mercurial treatment has now been definitely settled, since cases have been published in which the complication occurred where no treatment whatever had been followed. So far from giving rise to nephritis, mercury is one of the most powerful agents in its cure, though its use has to be watched. Thus, the inunction method is not a good one, on account of the difficulty of regulating the amount of the remedy that is absorbed. Mercurial stomatitis, which appears with great facility in patients whose kidneys are not in good order, is a good means of testing whether the drug is properly tolerated by the system. As a general thing, Van Swieten's solution at the dose of a tablespoonful *per diem* will prove sufficient, though hydrargyrum tannate, 0.10 to 0.20 (one and a half to three grains) *per diem*, can be given, on account of the efficacy of tannin in some forms of albuminuria.

Potassium iodide should always be associated with the mercury, and the dose should be raised as far as the digestion will tolerate,—forty-five to sixty grains a day. Gibert's well-known syrup is a good means of combining the two remedies, although the proportion of iodide it contains is too low.

In addition to the specific treatment, it is well to lay stress on the usefulness of the general treatment of the subacute nephritis,—absolute milk diet, stimulation of the skin, diuretics, light laxatives, etc.; while the frequent application of wet or dry cups to the lumbar region will also be found beneficial. It is particularly in cases of haematuria that the latter means is advisable.

The second form of syphilitic nephritis, which appears later on during the course of the infection, is essentially a chronic one, is often associated with other visceral lesions, and particularly with those concerning the liver, and is an important factor in syphilitic cachexia. In any case of chronic nephritis where there is at the same time hypertrophy of the liver and spleen, the specific origin of the disease should be suspected. This form of syphilitic nephritis is a very tardy

one; a case has been published in which it appeared in the eighteenth year after the chancre.

Unluckily in this variety, the gumma, which is the element amenable to the specific treatment, is uncommon. Thus, in sixty-three post-mortems of syphilitic nephritis, Wagner only found the gumma in three cases. Even when the gumma does exist, it is accompanied by the ordinary interstitial and parenchymatous process, on which even the most energetic treatment has little effect. In addition to this, waxy degeneration, so common in old cases of syphilis, is also a tenacious and often irremediable lesion. When the waxy degeneration predominates over the nephritic process, the important indications concerning *régime* that arise from this fact will be found further on.

In these tardy forms of nephritis potassium iodide is our great resource, whereas the indications for mercury are much more restricted. Some authors abstain from it altogether. At the best, its use is only advisable during convalescence, and after recovery from all serious tertiary visceral lesions, to prevent recurrence; but its action on albuminuria must be watched with great care.

Even when the state of cachexia, the hypertrophy of the liver and spleen, the diarrhoea, and the existence of chronic suppuration of the bones make waxy degeneration particularly probable, iodide can still be given with success, and a case published thirty-five years ago by Hérard has often been cited as an example of a therapeutic triumph under circumstances apparently desperate. The instance concerned a woman of fifty, who had already been successfully treated four years previously, but who had been reduced by a relapse to extreme cachexia. The liver was very large, and, in addition to general anasarca, there was enormous ascites and the urine contained quantities of albumen. Under the influence of potassium iodide recovery was prodigiously rapid, and the patient was seen in good health six months after her exit from the hospital.

In this form of nephritis with extreme cachexia, and particularly in waxy degeneration, exclusive milk diet is said to do more harm than good. All writers agree on the necessity of feeding up the patient. In addition to vegetables, old wine, meat, and eggs can be given freely. The syrup of iodide of iron and preparations of tannin and iodine can be alternated with the potassium iodide. Semmola's anti-Bright's disease draught deserves to be better known than it is:

R Potassium iodide, 1 grammme (15 grs.);
 Sodium phosphate, 2 grammes (80 grs.);
 Sodium chlorate, 6 grammes (90 grs.);
 Aquea destillatae, 1000 grammes (1 quart).

To be taken in twenty-four hours. The water can be replaced by an equal amount of milk, if preferred.

In such cases, even more so than in the *early* forms of syphilitic nephritis, great precautions are necessary after apparent recovery, as with these patients there is no question of *restitutio ad integrum*. The kidneys are atrophied and destroyed in certain parts, and improvement is only due to the hyperactivity of the physiological working of the parts left unharmed by the disease.

In the third principal form of syphilitic nephritis, the hereditary variety, the importance of the renal lesions, at any rate from a practical point of view, is more restricted. In two cases Lecorché and Calamon found small, granular kidneys, with enormous cardiac hypertrophy. Klebs and Parrot have cited undoubted cases of gumma. But, as a general thing, the lesions are latent, without any special symptoms, and are not distinguished from the other visceral manifestations and progressive cachexia.

Bradley, however, has published a case of recovery in a child of four months, with anasarca and albuminuria. The coexistence of cutaneous syphilis and disappearance of the albumen and anasarca in three weeks with the specific treatment rendered the nature of the nephritis unquestionable.

In such instances in infants the doses of mercury should be small. Parrot recommended Van Swieten's solution to be given in the following manner:

R Van Swieten's solution, 2 to 3 grammes (30-45 grains);
 Syrup of mucilage, 25 grammes (6½ drachms).

To be given during the twenty-four hours, in teaspoonful doses, before nursing.

Inunctions can also be prescribed, the doses for the following preparation being: two grammes, or thirty grains, up to three months of age; four grammes, or one drachm, from three to six months; and six grammes, or one and a half drachms, from six to twelve months.

R Mercurial ointment, 10 parts;
 Lard, 20 parts.

Tardy hereditary syphilis appears to affect the kidney very little. Still, Bartels published a case which he considers as having been complicated with waxy degeneration, and which recovered under the influence of potassium iodide.

In paroxysmic haemoglobinuria, syphilis, either hereditary or acquired, is a most important causal factor. It is said to be present in more than two-thirds of the cases. Recovery through the specific treatment is the rule, and potassium iodide particularly should be tried between the attacks, even when another cause besides syphilis appears to be to blame.

CYCLIC VOMITING.

READ BEFORE THE NEW JERSEY STATE MEDICAL SOCIETY, JUNE, 1899.

BY ALEXANDER MARCY, JR., M.D.,

Riverton, New Jersey.

MR. PRESIDENT AND GENTLEMEN,—In asking your attention for a short time to the subject of this paper, “Cyclic Vomiting in Children,” I offer no apology, for I believe it is a subject of very great importance, and one which is not well understood; nor has it had that attention which we would expect for so serious a malady. One reason why it has not received more attention is, I believe, on account of its comparative infrequency, and yet I also believe that many cases occur, the true nature of which are unrecognized.

The only reference to this disease in our text-books that I have been able to find is in Rotch’s “Pediatrics,” Holt’s “Diseases of Children,” and Keating’s “Cyclopædia of Diseases of Children.” Fenwick, in his work on “Disorders of Digestion in Infancy and Childhood,” speaks of recurrent catarrh of the stomach, and from his description of this disease, I suspect that it is probably a description of this same condition.

I have been fortunate, or, rather, I might say unfortunate, enough to have seen three cases during the past five years occurring in children, and while this disease may occur at any age, it is especially as to its occurring in children that I wish to confine my remarks.

One of these cases was of a mild type, the other two very severe. I will relate the history of these cases briefly, and will outline somewhat in detail a typical attack, as witnessed a number of times in each of the severe cases.

The history of the mild case was as follows:

CASE I.—B. R., American; female, aged six; well developed; apparently healthy. Had had the usual diseases of childhood. She

was taken with a severe attack of vomiting, coming on without apparent cause. I found the child very much exhausted from the almost constant emesis, which had been going on for nearly forty-eight hours. She had some fever, temperature 102° F.; slight pain and tenderness over stomach; tongue dry, very much coated; bowels constipated; urine scanty and highly colored; breath giving off a peculiar odor; respirations sighing and moderately slow.

I supposed I was dealing with a catarrhal irritation of the gastric mucous membrane, prescribed what I considered the appropriate remedies, and in a short time she was well.

Some months after this I was called to see her again, and found the same conditions, only somewhat more severe and lasting two or three days longer.

The next attack she had was very severe, and was accompanied with violent and almost constant choreiform movements of the muscles of the face, neck, and upper extremities. This attack was speedily relieved by a hypodermic injection of morphia and atropia.

One other attack, some time after this, also quickly yielded to the morphia and atropia, and she has remained well ever since.

CASE II.—A. H., born August 29, 1891; was a fine, healthy child at birth; weighed ten pounds; perfect, apparently, in every respect; was nursed at breast until fourteen months old. Up to this time he had never been sick, excepting an attack of whooping-cough when six months old. He was weaned from breast at fourteen months, and put on cow's milk, and as a result of this change in diet, together with the intense emotional disturbances incident thereto, he had a severe attack of gastro-enteritis, complicated with meningitis, which proved very serious. He made, however, a complete recovery from this illness, and continued perfectly well until June 12, 1893.

At this time he had his first seizure of vomiting. This was considered an attack of gastric irritation, due to acute milk infection, and after a few days he seemed perfectly well. He continued in good health until February, 1894, when he had another seizure, rather more severe than the first, from which, however, he recovered promptly. He had another slight attack in July of the same year, and then continued well until February, 1896. This seizure was more severe than any of the others, but complete recovery followed in a few days.

In March, 1896, he had measles, which was accompanied by an attack of vomiting, and two weeks after this he had another.

In May he had a very severe one, in which the symptoms became so very alarming that his recovery seemed very doubtful. He did recover, however, but in July he had another seizure, which was still more severe, if such a thing could be possible, and from which he recovered more slowly.

He continued well until October 8, when he was taken again, and although this time the disease seemed less severe than it had been before, so far as the vomiting was concerned, yet the evident depression of vital force, the bloody vomitus, the suppression of urine, the extreme exhaustion, all showed but too plainly the intensity of the toxæmia.

He died on the 9th, from heart failure.

There was an acute nephritis coincident with this attack, as shown by the presence of albumen, blood, and casts in the urine.

CASE III.—H. H., female, aged six years; was a strong, healthy, well-developed child at birth; was nursed at breast until eighteen months old. Development normal in every way. Had very little, if any, sickness until three and a half years of age. At this time she began to have attacks of vomiting, with fever. They were supposed to be due to indigestion; they came on suddenly, without apparent cause, lasted a short time, and recovery seemed prompt and complete.

In September, 1895, she had an attack which was very severe and which lasted one week. During the course of this she became so thoroughly exhausted that the physician in attendance thought her recovery impossible. She recovered, however, very rapidly, so that in a week after the vomiting ceased she seemed perfectly well. She continued well until August, 1897, when she was taken with another seizure, which was even more severe than the former, and which lasted eight days. Her condition was so alarming that her death was momentarily expected.

While in this condition she was seen by one of Philadelphia's most noted surgeons, who thought she could not recover. He was very positive that she had a diseased appendix, and that her vomiting was caused by it. In the event of her recovery he wished an opportunity of examining her again. She did recover, and after she had gotten perfectly well she was taken to him for more thorough exami-

nation. After very careful and painstaking efforts, it was pronounced chronic appendicitis, and operation for its removal was strongly urged.

The operation was performed and the appendix removed. Macroscopically it did not show any very marked evidences of disease. The microscopic study was as follows:

“Microscopic Appearance.”—The lumen of the appendix is in part occupied by a fecal concretion. There is considerable erosion of the mucous membrane. Mucous glands are here and there hyperplastic, the epithelial cells of the mucous glands are in great part the seat of mucoid degeneration, being occupied by clear, semitranslucent, semirefractive droplets. The lymph-follicles are hyperplastic and a few of the cells show slight retrograde metamorphosis in that they stain rather imperfectly; a few of the mucous glands also present similar changes. In the submucosa, and to less extent also in the muscularis and subserosa, there is a moderate amount of round-cell infiltration; the blood-vessels are distended and filled with blood, which presents an excessive number of leucocytes. The blood-vessel walls are thickened and exhibit considerable nuclei. The connective tissue of the submucosa is in great part homogeneous and contains relatively few nuclei.”

After her recovery from the operation, she seemed unusually well, gained in weight, had good digestion, regular bowel movements, perfectly clean tongue,—conditions which had not existed to the same degree before the appendix was removed.

This condition of perfect health continued for so long a time that I had about concluded that the removal of the appendix had, after all, solved the problem in her case. In March, however, ten months after the operation, she had another seizure, and died from exhaustion, after an illness lasting nine days.

As these severe attacks of cyclic vomiting are all so nearly alike, I shall simply attempt to describe a typical one, somewhat in detail, in order that you may get a faint idea of the severity of the disease, and even then, though I might be able to picture it graphically, you will hardly realize what it means, and I am sure, until after you have actually seen it, and watched it closely from hour to hour as it goes through its various stages, you will have but a very imperfect idea of what it really is.

The first noticeable symptom is loss of appetite, usually a refusal

to eat the evening meal; the child seems a little more tired than usual, and wants to get to bed; if the observation is made, the tongue will be found slightly coated, the breath a little heavy; the bowels, perhaps, have not moved that day; the urine has increased in quantity; no fever. The sleep is a little more restless than usual. During the night, usually between twelve and two, the child will awaken and complain of feeling sick, and in a very short time begins to vomit. The first matter vomited will be the contents of the stomach, which will consist of particles of food from the day before, with larger quantities of mucus.

There will be some pain around the umbilicus, and a very pronounced aversion to being examined. Sometimes there will be two or three loose movements from the bowels during the next few hours, with a copious flow of urine. Usually, however, the bowels are constipated, even obstinately so. The temperature at this time becomes somewhat elevated, and the child grows very restless.

The disease now is well under way, and every few moments the child is vomiting. After the stomach has been emptied of its contents, the vomitus consists of mucus, then mucus stained with bile, and finally a peculiar bronze-colored mucus, occasionally containing some blood. Oftentimes there is violent retching, and nothing is ejected. There is intense thirst, and the little patient will beg for water, milk, champagne, etc., and vomit any or all of them as soon as they are swallowed.

Anything and everything that is taken into the stomach is almost immediately rejected. At this time there is noticed a peculiar odor given off by the vomited matter, as well as by the breath. This is so characteristic that I could diagnose a case of cyclic vomiting from that alone.

The temperature is now perhaps 103° F., the pulse 140 to 160, the respirations irregular and sighing, the urine very much diminished, and peristalsis entirely suspended. The tongue is very much coated, the lips dry and parched, the belly flat, the entire body shrunken in appearance, muscles flabby, skin harsh and dry. You might suppose the child had been ill weeks instead of hours. There is also at this time great restlessness. The patient goes from one side of the bed to the other, from the head to the foot, rarely still ten minutes at a time. There is practically no sleep. There is a curious intolerance of bedclothing, and not even a sheet will be permitted to

remain over him. Although he may be in a sound sleep, the moment anything is placed over him, he will awaken immediately.

This has continued for perhaps forty-eight hours, and the patient is becoming very much exhausted. The pulse is getting alarmingly rapid and weak; the temperature is falling, until it becomes sub-normal; the eyes are half closed, with balls partly rolled up, the conjunctiva injected, the eyes sunken in their sockets. The whole picture is ghastly. The condition of the patient is intensely critical, the physician's anxiety extreme. Still it continues for seventy-two hours, the conditions getting worse. Now the extremities are cold and blue, the peripheral capillary circulation practically suspended. The pulse gets weaker and more rapid, until it becomes impossible to count it. The temperature begins to go up, and in a short time you will find it 105° F. in the rectum. The respirations get more and more irregular. The restlessness is less pronounced, on account of the extreme weakness.

It is now four or five days since the attack began, and you have given up all hope of the patient's recovery. You calmly and sorrowfully await the end, hoping that it may not be far away. At this time you notice that the intervals between the vomiting are longer, and that the little patient seems inclined to sleep somewhat. This becomes more and more evident, until the child finally passes into a profound state of somnolence. This may continue for hours. During this sleep the temperature falls, the hands and feet grow warm, the pulse becomes stronger and less rapid, and when the child awakes you can see very plainly that the crisis has been passed,—the attack is over. The convalescence is marvellously rapid, and in less than a week you would not know that there had been a severe illness at all.

In two of my cases, however, the result was different, although they had both had attacks just as severe as that I have attempted to describe, and recovered. Yet there finally came one from which the patient did not recover.

Now, what is the nature of this disease, and what its cause? There is very little literature on the subject. A few cases have been reported in the medical journals, and it is mentioned in a few only of the many text-books on the diseases of children. Holt, in his work on "Diseases of Infancy and Childhood," says,—

"In cyclic vomiting it is quite probable that the cause is the

accumulation of some toxic agent in the blood, and that the vomiting may be eliminative."

He also says,—

"This condition is one which has received but little attention, being classed by some as a *gastric neurosis*. While at the present time we are not in a position to give it a definite pathology, it seems to be associated with a general derangement of nutrition, which is in some way connected with formation and excretion of uric acid, but it is not certain that all cases have the same origin."

Rotch, in his "Pediatrics," says,—

"There is no name which can be given to this disease except that of *persistent vomiting*, as no single definite cause nor any pathological lesion has ever been found to produce it. It has not even been shown that it is a primary disturbance of the stomach. In fact, in many cases it is possible that the source of irritation is entirely outside of the stomach, and perhaps connected with the great sympathetic ganglia, such as the solar plexus.

"The inciting cause of the vomiting in most cases is obscure, but it is evidently varied. It does not seem to be produced especially by errors of diet, but, on the contrary, occurs in children where diet has been most carefully regulated. Undue exposure to cold, fright, and excitement, all appear to me to have sometimes an etiological influence on the disease. This form of vomiting may occur at any age. I have met with cases in young infants and throughout the whole period of childhood. The attacks may occur not only in delicate, nervous children, but also in those who are quite vigorous."

Pepper, in his article on "Cyclical Vomiting," in Keating's "Cyclopædia of Diseases of Children," says,—

"That this disease is not primarily dependent upon diseases of the stomach, either organic or functional, I am convinced, though observers as able as Fenwick describe it as a recurrent catarrh.

"The entire absence of gastric symptoms between the recurrences, and the frequent lack of any reasons for accepting the existence of an inflamed mucous membrane during the attacks, together with the common evidences of derangement of the nervous functions, are to me sufficient proof of its dependence upon influences beyond the stomach, though what these influences are cannot be stated.

"Some cases which have been published under this title, may,

perhaps, belong to another class of neurosis of the stomach; they are not, improbably, examples of periodic hypersecretion.

"There are others, however, of which I have seen several examples, in which there was apparently no abnormality of secretion. For these cases we have no adequate explanation.

"Nervous depression is the rule in the subjects of this affection, and exposure to cold, emotion, or other well-defined causes may bring on attacks, but in what way the result is produced is not known. Leyden's original explanation of the condition as a neurosis of the vagus is attractive, but leaves us in want of knowledge of the nature of the neurosis.

"The vomiting may be eliminative of some ptomaine recently produced or accumulating in the system, and the increased toxicity of the urine, observed by Hunter in one of Holt's cases, is an important support of this view, but the origin of such a possible ptomaine is unknown."

My own belief, after a very careful study of these cases, is that they are examples of auto-intoxication due to ptomaine-poisoning; that this ptomaine is formed in the intestinal canal, and is absorbed into the blood, producing a toxæmia. The vomiting is produced primarily by the effect of this poison on the nerve-centres. The poison is eliminated by the mucous membranes and the skin. The catarrhal inflammation of the stomach, bowels, and kidneys is a secondary process, and is not present excepting in the severe type of cases.

In support of these views, let me call your attention briefly to some important facts as obtained in the history of the cases that I have seen.

1. A study of the urine before an attack shows that it is always increased in quantity, of high specific gravity, very acid, and containing large amounts of indican. The uric acid, while not in excess, is rarely much below the normal.

2. During the first few hours of the disease there is but little change in this respect, but gradually the quantity of urine diminishes, the indican disappears, and acetone in large quantities is found. Acetone is also given off by the breath, and causes a peculiar odor, which is almost pathognomonic.

This evidence bears strongly on our first point, that there is auto-intoxication due to ptomaine-poisoning.

The post-mortem examination of the second fatal case gives us

some positive data from which we can derive some of our conclusions.

The examination was made twenty-four hours after death, and the following notes made:

Body of female child, extremely emaciated. Rigor mortis well marked. Thorax opened and revealed well-developed heart and lungs, there being marked hypostatic congestion of the latter. Abdomen opened; stomach found empty, the mucosa showing marked changes, due to inflammatory action. This was especially noticeable at the pyloric extremity. Large areas showed extravasated blood under the mucosa, with apparent softening. This condition was also marked in the duodenum, in the jejunum, and in the ileum, especially at the ileo-cæcal valve, the caput coli, and the ascending colon.

The glands of the intestinal canal were very prominent, Peyer's patches being particularly noticeable. Blood extravasations were also prominently noticeable here.

The liver was normal in size, somewhat altered in color, and appeared to be fatty.

The gall-bladder was *distended with bile*.

The spleen, pancreas, and kidneys were apparently unaltered.

The microscopical appearances are as follows,—this being a copy of the report of Dr. A. O. J. Kelly, the pathologist who examined the specimens:

“Stomach and Intestines.—There is almost complete necrosis of the entire mucous membrane of both the stomach and intestine. Most of the epithelial cells do not stain at all, they being represented by an indistinct granular mass of tissue. A few of them, however, reveal indistinct nuclei, which are without sharp demarcation. In a few places the deeper cells are not so intensely necrosed as the superficial, but in other regions, on the other hand, the necrosis appears to have invaded the submucous coat. There are slight proliferative changes in the submucous and muscular coats.

“The Liver.—Microscopically the hepatic lobules are seen to be the seat of a great collection of clear vacuoles, which are sharply demarcated, and roundish or oval in shape. They are distributed uniformly throughout the acini, are situated within the liver cells, and vary considerably in size. Many of them are very small; others as large or larger than a normal liver cell. Many of the larger ones have evidently resulted from the coalescence of several smaller ones.

Corresponding to these in all respects, save that they are translucent and highly refractive, there are a few bodies, very evidently fat. It is fair to assume that the vacuoles have resulted from the extraction of the fat during the processes of preparing for histologic study. The liver cells are distorted in accordance with the size and shape of the contained fat droplets or vacuoles, their protoplasm severely compressed, but still revealing its normal granular appearance. The nuclei of the liver cells are displaced, but stain well. In a very few situations the interlobular connective tissue appears the seat of some slight proliferation.

“The Pancreas.”—In some of the lobules the outlines of the cells are indistinct; the protoplasm stains diffusely and is hazy; the nuclei stain less than normally and not so sharply. Otherwise there are no deviations from the normal.

“The Spleen.”—There is considerable distention of the blood-spaces and here and there more or less diffuse hemorrhagic infiltration. In other situations there are some blood-pigment granules between the splenic cells. In some situations also many of the pulp-cells are indistinct in outline, their nuclei stain indistinctly and not sharply, and the protoplasm stains diffusely and is hazy.

“The Kidney.”—The lining cells of some of the Bowman capsules are proliferated, the capsules rather fuller than normal, with proliferated endothelial or other cells. A few of the cells lining the convoluted uriniferous tubules reveal indistinct nuclei, and their protoplasm appears abnormally granular. Otherwise there are no deviations from the normal.

“Anatomical Diagnosis.”—Necrosis of the mucous membrane of the stomach and intestine; fatty infiltration of the liver; slight parenchymatous degeneration of the pancreas, spleen, and kidney. In view of the fact that the patient had been dead less than a day when the necropsy was performed, it is not likely that the extensive necrosis of the mucous membrane of the gastro-intestinal tract is a post-mortem change.

“A. O. J. KELLY.”

These changes undoubtedly show a very severe gastro-intestinal inflammation. The elevated temperature of the third or fourth day and the pain and tenderness to the touch point plainly to an acute inflammatory process.

The first fatal case was perfectly well up to the time of the beginning of the vomiting, which was about ten P.M. There had been found indican in large amounts in the urine for a week before; in twelve hours after the attack began the child was vomiting blood, had high temperature, and was completely prostrated, although the number of times the vomiting occurred was less than in previous attacks. In twenty-four hours the urine showed albumen, casts, and blood, and shortly became entirely suppressed, and in forty-eight hours death occurred, from heart failure. The pulse grew weaker and weaker, until it could not be felt at the wrist. The heart continued to beat for several hours after this, finally stopping in diastole. Unfortunately no post-mortem was made in this case, but death was undoubtedly caused by the intensity of the toxæmia, as sufficient time had not elapsed for exhaustion to occur.

There is a curious neurotic element in these cases, as shown by the extreme restlessness, the absolute intolerance of bedclothing, the cold and dusky extremities. There is also an intense pruritus after the attack, which I think indicates an attempt at elimination by the skin.

The conditions which provoke an attack are varied, but are possibly due, for the most part, to the ingestion of certain kinds of food, coupled with an imperfect intestinal digestion and lowered vital force. Whether the biliary secretions influence the conditions in any way I am unable to say, but a very noticeable fact observed at the post-mortem was a distended gall-bladder. As yet I have not completed my study of the contained secretions, and cannot say positively that there is anything abnormal about them, but I suspect that there is.

Treatment in these cases is practically useless. The disease is self-limited and tends to recovery, but oftentimes the patient is exhausted before nature can eliminate all the poison.

The more you put in the stomach, the more the vomiting.

Predigested food, sedatives, and stimulants by the rectum are of doubtful utility, but should always be employed. Hot baths and external heat give some comfort, and help the skin to eliminate. A small blister applied to the epigastrium sometimes serves to do a little good.

Hypodermic injections of morphia and atropia do control the vomiting some, relieve pain, and tend to produce sleep.

Hypodermic injections of strychnia stimulate the respiratory centre and tone up the nervous system.

Hypodermic injections of tincture of strophanthus or digitalis strengthen the heart.

Normal saline solution, by transfusion, by hypodermoclysis or enteroclysis offer, I think, the greatest hope of success in the severe type of cases, and I think it should be employed very early after the stomach and bowels have been thoroughly cleansed of any retained undigested food or fecal matter.

ANCHYLOSTOMIASIS AND GRAVES'S DISEASE.¹

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF BONN.

BY PROFESSOR FRIEDRICH SCHULTZE,

Professor of Special Pathology and Therapy and Director of the Medical Clinic at
the University of Bonn, Germany.

GENTLEMEN,—I congratulate you on the number which I find present to meet me this morning at the opening clinic of the semester. For my colleagues the Seven Mountains² are wont to hold forth in most tempting fashion on such a beautiful day in the spring-time as this. I compliment you on your self-denial, however, and am glad that you are learning so well and so soon the lesson that every medical man must take to heart, that his personal convenience and pleasure must ever give way to the duties of his noble profession. It is a lesson that is apt to be neglected in our money-making day, but it is well worth the learning, since it lifts the profession, with its magnificent possibilities in a humanitarian way, above the sphere of the mere craftsman or tradesman.

And now I am glad, since you are here at the price of a little self-sacrifice, that I can reward it by presenting to you a case that I think you will agree with me is extremely interesting. Our patient is a young man of twenty-one, who came into the hospital four days ago complaining of headache and an intense tired feeling. This was more than merely a tired feeling: it was an absolute state of exhaustion, which had prevented him from working for some time.

He told us, too, that for some months past he had become very pale, though when you look at him you see at once that there was no need of his telling us, since it is almost painfully evident. You see how intense the pallor is. There is not the slightest bit of color left

¹ Reported by James J. Walsh, Ph.D., M.D.

² The Seven Mountains (Die Siebengebirge) contain the most beautiful of all the Rhine scenery. They are not far from Bonn, and may easily be seen from the clinic windows.

in his mucous membranes even. His conjunctivæ are almost absolutely colorless. The absence of all hint of blood-coloring matter gives a shade of grayish yellow to the skin.

Here we have just the type of case that would seem to lend itself easily to symptomatic treatment. We have headache, that can be treated with one of the newer coal-tar derivatives, with the hope that the anæmia to which it is due will improve under general treatment. The anæmia indicates that iron is needed; the tired feeling, that the patient needs tonic stimulant treatment, until the anæmia gets better under the use of iron, when that too will disappear. That would seem to some doctors an eminently well-reasoned set of therapeutic indications, and they might even compliment themselves on the way in which their therapeutic scheme held together. I need not say that nothing could be more false, or more superficial, or less scientific.

Anæmia is evidently at the bottom of all the symptoms, but what is its cause? This we must get at if we would really hope to benefit our patient. What are the usual causes of anæmia? First, of course, lack of blood due to its loss by hemorrhage. Frequent or prolonged epistaxis, hæmatemesis, or pulmonary hemorrhage must be at once thought of, when there is no history of trauma in anæmia. Inquiry suffices for these, since they cannot occur without the knowledge of the patient. Hemorrhage from the rectum may, however, occur in small and oft-repeated amounts without the patient's knowledge, or may attract his attention so little that he will fail to mention it to his physician unless particularly asked about it. In all cases of anæmia a rectal examination is advisable, unless some other cause for the bloodlessness immediately suggests itself. I need scarcely add here that in women, of course, careful inquiry is to be made as to hemorrhage from the genitalia.

Besides loss of blood, the loss of any of the body fluids may provoke anæmia. Chronic diarrœa, for instance, by withdrawing serum from the circulation, may cause serious anæmia. Lactation acts in a similar manner when excessive or too prolonged, and these terms are extremely relative. One woman is not affected by the strain of twelve months' lactation, while another succumbs to two or three, and yet there may be nothing in the external appearance of either to indicate the reason for the vigor or the weakness of the blood-making organs.

Then there are a series of causes which are twofold in character:

First, they withdraw serum from the circulation; second, they permit of the entrance into the circulation of toxines that still further disturb blood composition and equilibrium. Purulent and tubercular processes and kidney affections are the types of what I refer to. The loss of fluid in pus is sometimes considerable; the absorption of bacterial toxines not only disturbs the composition of the blood itself, but palsies trophic influence and inhibits cellular energy in the blood-making organs. In kidney affections the withdrawal of albumen has its influence in lessening the nutritive qualities of the blood in the circulation itself; toxines that should be excreted are retained and act as depressants on all function, while the fluid withdrawn from the circulation in oedema and in various dropsical effusions is not without its effect in disturbing blood equilibrium.

Somewhat analogous in their effect are rapidly growing tumors, especially those which are called malignant. The amount of nutritive substance required for the carrying on of the extremely luxuriant cellular activity which they so often exhibit is in itself frequently a serious drain upon the system. Besides, though we are as yet so much in the dark as to their cause, it can scarcely be doubted that their growth involves the introduction into the general circulation of substances that act as depressants upon all tissue activity. This makes its appearance earliest and is most noticeable in the anæmia that develops in cases of cancer, and is the most striking symptom of the cancerous cachexia.

With none of these things have we to do here. There is no purulent or tuberculous process present. Kidney trouble there is none. His urine is normal in amount and specific gravity and contains no casts. The slight amounts of albumen which it contains are evidently due to the anæmia. Either the thinner hydræmic blood permits the plasma to part with its albumen more readily, or the cells of the kidney tubules have suffered in their nutrition because of the anæmia and permit an exosmosis from the blood of substances which they would otherwise retain. Our patient is almost too young to have a malignant tumor, and there are absolutely no signs that point to the presence of one.

There remain to be considered certain not well understood conditions of the blood-making organs, which produce some form of anæmia or oligæmia as a symptom. The anæmia in our patient, it may be said at once, is too marked to be due to leukæmia. There is in that

disease, corresponding to the pathological increase in the number of white blood-corpuses, not only a relative but an absolute decrease in the number of reds and in their haemoglobin contents, but that never goes so far as to produce the intense pallor noted here. Besides the examination of the blood gives us no increase in the number of leucocytes present.

The blood-picture is not unlike that presented in chlorosis, and the general appearance of the patient would support that view. While delicate, he is not extremely emaciated, and there is a tinge perhaps of yellowish green in his peculiar paleness, and *χλωρός*, after all, means yellowish green. The fact that our patient is a male is not enough of itself to preclude the diagnosis of chlorosis, for, while it is characteristically a disease of young women, it is not exclusively so. We have learned of late to group certain analogous anaemic conditions which occur in young men about the age of puberty under the head of chlorosis.

His age is, however, against that diagnosis: he is past nineteen. He has not had anaemia for years, now better and now worse, with chlorotic relapses, which may occur in young men as well as in young women, but has been affected only for some months. Besides, the diagnosis of chlorosis is only the giving of a name to a group of symptoms; it tells us nothing of the nature of the disease, does not help us to a rational therapy by leading us to the cause of the disease, and is only to be thought of when no other can be found. It would be eminently unsatisfactory to rest our case here with the diagnosis of chlorosis.

There remains to be considered, of the so-called essential anaemias, the form known as progressive pernicious anaemia. We have in the blood-picture in this case, in the seemingly progressive character and in the intensity of the blood disturbance, most of the symptoms on which a diagnosis of progressive pernicious anaemia could be made,—in fact, only a few years ago would have been made. We have learned of late, however, that most of the cases that used to be called progressive pernicious anaemia are really not primary but secondary anaemias, the cause of which remains hidden for a time. Among the obscure causes are especially malignant tumors of bone, atrophic gastro-intestinal conditions, and intestinal parasites of several kinds.

Of late years the etiological connection between various forms of intestinal parasites and severe anaemia has attracted a great deal of

attention. They act mainly by the abstraction of blood and of albuminous substances from the system, though it seems clear, too, that they either themselves produce, by their metabolism, toxines which are absorbed and have a hæmolytic effect, or their presence so disturbs the proper preparation of the chyme that such hæmolytic substances come to be absorbed in the shape of unsuitable albumoses. A number of intestinal parasites have been held accountable for such effects. The anchylostomum duodenale and the bothryocephalus latus undoubtedly do produce them. There are well-grounded suspicions that the tricocephalus dispar may cause anæmia, and of late the various tapeworms, and even lumbricoides, the ordinary round worms, have seemed to some to be the active agent in severe anæmias, especially in young people.

That the anchylostomum duodenale is the cause of those severe anæmias which resemble pernicious anæmia in every particular, even in being fatally progressive unless their cause is speedily removed, has been known for some time. It was first noted in Europe among the Italian workmen in the St. Gothard Tunnel. Afterwards here in the Rheinland it was found as the cause of severe anæmias among the workers in brick-yards. Menche, here at Bonn, first demonstrated the parasite and its eggs in the stools of patients who had been working in a neighboring brick-yard. As our young patient is a brick-maker, it was at once suspected that the anchylostomum might be the cause of the anæmia in his case, and the eggs of the parasite were looked for in his stools. They occur in immense numbers, and so were easily found, and then the diagnosis was made. A diagrammatic sketch of the eggs of anchylostomum you have here, and you may see them for yourself under the microscope. We at once gave our patient a dose of the vermifuge usually employed for this parasite, *filix mas*, and so I am able to show you a number of the parasites also.

The anchylostoma are not as numerous in this case as they have often been found. We have been able to find so far only one hundred and twenty-seven worms, while as many as three thousand have been recovered from the stools in some cases. That is the maximum, I think, so far. The number of eggs produced by the female is very large, so that a great quantity of albumen is extracted from the organism of the host for their production. In the dish that I pass around are both male and female parasites. The thicker, longer, rounder ones are the females; the thinner, shorter, more thread-like

organisms are the males or virgin females. The males, you will notice, have a curve at the end, while the females are without it. As the number of males here seems to exceed that of the females, while usually there are more than twice as many females, the conviction that we have not yet gotten all the parasites is strengthened. We shall carefully examine the faeces for more eggs, and if they are found we shall after a few days give another course of *filix mas*.

So far we have occupied ourselves very little with the patient himself. We know his history; we have examined his blood and his stools. We have undoubtedly found the cause of his present symptoms. We might remove this cause and think our duty fulfilled. I cannot warn you too strenuously against this incomplete and unscientific way of handling patients. The prognosis of a case and the progress and complications that may be looked for in many cases depend not on the principal affection and its cause, but on the general condition of the patient, and on minor affections or congenital or acquired conditions which may be present practically without having attracted the patient's attention. Our present case furnishes an excellent and interesting example of this; so I insist on it.

Examination of our patient's pulse shows that it is quite rapid. Just at present it is about 120, but it has been even higher in the wards. It is soft and regular and easily compressed. When we uncover his thorax we are struck by the extent of the heart's impulse. It can be seen in the second, third, and fourth intercostal spaces, and there is even distinct pulsation here in the hollow above the sternum. This regular rapidity of the heart-beats—tachycardia, as it is called—might be due to the intense anaemia, but if that were the only cause we might expect irregular rapidity. The heart's impulse—for we can scarcely call this fluttering impulse of the whole heart against the chest wall the apex-beat—would not then be so broad and noticeable. It would be weaker, often scarcely visible, and would occupy about the position of the normal heart-beat.

There is nothing the matter with the heart itself to account for the tachycardia. There is slight dilatation, demonstrable on percussion, but that is only partly perhaps real dilatation from the poor nutritional condition of the heart muscle, and is mostly due to the fact that the lungs are not much distended with air, since very little exercise is being taken, and so more of the heart is uncovered by lung. There is a soft, blowing, systolic murmur to be heard, but this is so

common in anaemic conditions that it means nothing, and certainly cannot for a moment be thought to indicate an affection of the endocardium with involvement of the valves. When you look for abnormal pulsations in the cervical region, you notice that his neck is fuller than normal and that this fulness occupies about the position of the thyroid gland. To palpation the tumor is not hard, but soft and compressible, though it may easily be defined; it communicates no thrill to the examining finger.

With two of the symptoms of Basedow's or Graves's disease, tachycardia and enlargement in the thyroid region, we naturally look for the third of the triad of symptoms characteristic of the disease,—the exophthalmos. There is no doubt that when you look at the eyes, knowing the existence of the other symptoms, you find that they are more prominent than usual, but this prominence is but very slight. When asked to open his eyes wide, you notice a wider band of white above his pupil than is normal, and even after he has allowed his lids to relax you notice the sclera slightly exposed above the cornea. This indicates at least a slight degree of exophthalmos. It may, however, be congenital. I have, in fact, asked him to look carefully in the mirror and tell us if he thinks his eyes are more prominent than they used to be, but he says that he thinks not. None of his friends have remarked any such change in him. Here in this part of Germany, too, we dare not place much weight on the existence of slight struma as a symptom of Basedow's disease. In many parts hereabout goitre is endemic. Our patient comes from Reith, near Cologne, and he tells us that a number of people in his native village have these enlarged necks. So, though we have the characteristic triad of symptoms, we cannot absolutely make the diagnosis Basedow's disease on the strength of them.

There is one very important confirmatory symptom: it is the tremor that Marie pointed out some years ago as so characteristic of the disease. When I ask him to hold out his hand with the palm down and the fingers slightly spread, you notice the slight continuous tremor, a series of fine vibrations, regular and rhythmical. He has not the other symptoms that are sometimes noted in Basedow's disease,—Stelwag's symptom, for example, the failure to close the eyelids over the eyeball involuntarily at short intervals, as all normal people do. If you have been watching him carefully, you will have noticed that, even during our last sentence or two he has winked

several times. Von Graefe's symptom—the failure of the upper lid to follow the eyeball beyond a certain distance when the patient is asked to look downward—is also not present. Both of these symptoms are, however, usually associated with marked exophthalmos, and, as this is not present in our case, their absence in our patient is not surprising.

It is impossible to say definitely whether we have here an incipient case of Basedow's disease or not, though it is very probable that we have. Basedow's disease developing on the groundwork of the severe anaemia of ancylostomiasis is extremely interesting. It is probable, too, that, if there is here at present only a larval form of the disease, we can look for the more outspoken development of the symptoms within the next few weeks, for the severe anaemia due to the presence of the ancylostomum does not improve at once on the removal of the parasite, as the exhausted blood-making organs require some time to renew their perfect activity; and, if the theory of the absorption of toxines from the parasites as a partial cause of the anaemia be accepted, and it seems very probable, it requires some time also for the elimination of these toxic products from the tissues. In a run-down condition like this the symptoms of incipient Basedow's disease should develop even more rapidly than usual into the outspoken stages of the disease; so it is probable that at a future clinic we shall be able to demonstrate more definitely the existence or non-existence of that disease.

The patient's history does not throw much light on his case, except for his occupation. He has been a brickmaker now for six years, though he did not work at his trade from 1895 to 1897, so that the infection is a recent one. He has always had a good appetite and no stomach or intestinal trouble to speak of. When examined a little more closely he confesses to an occasional diarrhoea of late. This has probably been due to the irritation of the presence of the parasite and its products in the intestinal canal.

He says he looked for blood more than once in the diarrhoeic stools, but there was none. It is usually said that shortly after infection with ancylostomum traces of blood are to be found in the stools. In fact, it has sometimes been considered an almost pathognomonic sign of the invasion of the parasite in people exposed to infection. We have not found this claim substantiated here at Bonn, though we have had a number of cases under observation. It frequently seems

to happen that cases run their course without a trace of intestinal hemorrhage, making itself noticeable by any external sign, though the cases are very severe.

The prognosis of our case here is not bad, though, of course, it is seriously modified by the presence of this larval form of Basedow's disease, the chronicity of which is well known, as well as its tendency, even when uncomplicated, to produce a cachexic anaemia. In ancylostomiasis it is very important to discover the parasite as early as possible, since there are in the literature a certain number of fatal cases, in which patients succumbed to a progressive anaemia, pernicious in that sense at least, even after the removal of the parasites, the blood-making organs seemingly having been exhausted by previous compensatory over-exertion. Where a patient comes to your office, the obtaining of a stool may be a matter of considerable inconvenience, sometimes of delay. In such cases a cotton tampon may be inserted into the rectum and withdrawn, when the eggs of the parasite, usually plentifully present in the rectal ampulla and just above the sphincter ani, will be found on its surface. Of course the microscope is required for their detection.

For treatment we gave our patient, after a preliminary purgative, two drachms (eight grammes) of the ethereal extract of male fern, to be taken at one dose. To robust adults we give two and a half drachms (about ten grammes). To insure its success, the extract should be freshly made, and in this fact may, I believe, be found the principal reason for the reported failures with it. Another reason for failure seems to be that the parasites are protected by mucus in the intestine. They seem to become enveloped in mucous material through which the vermifuge or parasiticide is unable to penetrate. As their presence causes by irritation an excessive secretion of mucus, the preliminary purgative is always advisable for its removal. Sometimes this mucous catarrh of the intestine constitutes a condition itself requiring treatment. In all cases, however, where ancylostomum eggs have been found and yet the administration of *filix mas* fails to bring away parasites, or causes the expulsion of only a few, the character of the stools should be carefully looked to, for in them will often be found the cause of the supposed idiosyncrasy of the parasite in certain cases. About three hours after the *filix mas* another purgative should be given; we always use castor oil here, as the gentlest and most efficient.

Where this fails to eradicate the parasite, which, as already said, may be found out by re-examination after a few days of the stools for parasitic ova, another and slightly larger dose of *filix mas*, preceded by more thorough purgation, may be tried. It is to be remembered, however, that the patients are, as a rule, in a state of intense anaemia, due to the parasite, and are by no means able to stand drastic purgation. Besides, it must not be forgotten that *filix mas* is an active poison, which is in fact the reason why it acts as a parasiticide in these cases, and the dose must never be much larger than that suggested. Certain patients, too, have an idiosyncrasy for *filix mas*, and present toxic symptoms from it very easily; so that until the patient's reaction to the drug is known, a dose larger than that we have suggested should *never* be given.

Where a second dose of *filix mas* fails effectually to remove the parasite,—though this failure must not be inferred from the patient's anaemia, which will continue for some time, but from the presence of the ova in the stools,—thymol, which is often effective in such cases, may be tried. Four or five grammes of thymol (one drachm or a drachm and a quarter) may be given. It is easiest taken in a little wine, some of our stronger Rhine wines, or in a little cognac, or, if preferred, in beer.

ACUTE FORMATION OF GAS IN THE STOMACH,— TWO TYPES AND A THEORY.

BY A. L. BENEDICT, A.M., M.D.,

**Professor of Physiology and Digestive Diseases, Dental Department, University of
Buffalo; Special Consultant in Medicine, Buffalo Hospital of the Sisters of
Charity, Buffalo, New York.**

AMONG the causes of formation of gas in the stomach, or its issue from the stomach, several factors must be included. In swallowing, not only is the larynx closed by the epiglottis, but the posterior nares are closed by the muscles of the soft palate, so that the pharynx is a practically air-tight cavity except for the oesophagus. Thus, the muscular acts of deglutition force more or less air into the stomach along with the bolus of food or the drink. Usually no more air is swallowed than can be disposed of by absorption or by passage through the pylorus, but habits of hasty eating and, perhaps, individual anatomic peculiarities of the pharynx may render the amount of air swallowed considerable. If the cardia is relaxed, there follows a postprandial belching which is disagreeable and mortifying, but not pathologic, and which is amenable merely to the practical suggestion to eat more slowly and more carefully.

The belching of gas after the ingestion of carbonated or aërated waters and of rapidly fermenting food needs only to be mentioned.

The commonest kind of gas-formation in the stomach is that produced by an excess of bacteria and yeasts, favored by one or both of two frequent abnormalities, deficiency of hydrochloric acid and stagnation of stomach contents, either on account of organic obstruction at the pylorus or of motor weakness. Extreme stagnation is apt to cause gas-production even in the presence of considerable quantities of hydrochloric acid, while good motor power and relative sterility of ingesta may allow a prolonged deficiency of hydrochloric acid without much organic fermentation. It is unnecessary to refer at length

to this form of gastric flatulence, especially as it is a symptom or rather condition common to many different diseases.

I wish, particularly, to describe two rather uncommon types of gastric flatulence, the first with infrequent belching, the second mainly characterized by belching. Both of these types are apt to become somewhat serious of themselves, without regard to the underlying cause. It is obviously impossible actually to present illustrative cases at a clinic.

As an illustration of the first type may be cited the case of a man of thirty, who had led a comfortable life, and whose habits for two or three years had been excellent, though he had formerly used alcoholics rather freely and had used tobacco considerably in both ways. He had had dyspepsia for several years and had formerly vomited frequently, especially after drinking beer, but lately he had been unable to vomit, though often nauseated. Right here I may say that, contrary to what might be supposed, comparatively few of my patients give a history of vomiting, the latter being essentially a conservative process which guards the stomach and intestine from sources of irritation and disease. The salol test resulted positively, after extracting with ether, half an hour after the meal; and three-quarters of an hour after the meal without using ether. The potassium iodide test resulted positively after fifty minutes, but as a control capsule did not dissolve in water within an hour, the test was really one of digestion, not of absorption. Personally, I do not regard these tests as of the least practical value. I concede that they may be significant when exceptional results, well beyond normal limits and controlled by other tests, are obtained. But extreme degrees of motor disturbance or absorptive delay are more easily diagnosed by simpler means. The patient had some rotary spinal curvature and a long thorax, but presented no visceral abnormality on physical examination, though he was sure that he had heart-disease. However, his hand trembled, he gave a distinct history of cardiac palpitation, and he was in a highly nervous state.

After about six weeks' observation and treatment, during which he seemed to improve, he came to the office at midnight in a state of great alarm, suffering from an attack such as he had already described as occurring before I saw him. He was awakened from sleep by a feeling of suffocation which was relieved temporarily by raising considerable quantities of gas from the stomach. Although he com-

plained of a feeling of cardiac oppression, the pulse was fairly strong and regular. Still the distention of the stomach was extreme, corresponding by auscultatory percussion to the limits of the inflated stomach shown in anatomies, and which are not found under normal conditions in life, and it seemed advisable to evacuate the gas through a stomach-tube. The tube was withdrawn shortly because of the nervous condition of the patient, but the spasm of the cardia was sufficiently relaxed so that he continued to gulp up large quantities of gas having the odor of vinegar. Soda and small doses of carbolic acid were administered, and relief soon followed. Two days later hydrochloric acid was prescribed regularly, to check the tendency to fermentation. No further attack occurred during my observation of the patient, but he left town soon afterwards, and I have heard that he still suffers from "heart-disease" and that he has occasional attacks of nocturnal ballooning of the stomach.

The possible danger of this type of gas-formation depends on spasm of the cardia. Milder forms, in which the cardia relaxes often enough to allow spontaneous evacuation of the gas, are very common, especially in lithæmic patients who describe themselves as animated vinegar- or gas-factories. While temporary relief is obtained from using soda, I must remind you that this does not fulfil any indication for radical treatment, and that hydrochloric acid is usually needed. While I have seen a number of cases of ballooning of the stomach sufficient to produce cardiac embarrassment and great terror on the part of the patient, and while even a mild attack occurring in a stomach already the seat of an ulcer is well known to be a cause of fresh hemorrhage and of perforation, I have not personally seen a case in which it seemed to me that the immediate danger was more than theoretic.

The second case, at first thought, seemed to me to be essentially similar to the first, but it is to be sharply differentiated and to be differently treated, though you may not accept the theory which I am going to set forth as an explanation. The patient was a married woman, aged forty, in comfortable circumstances, and enjoying fair general health. She gave no history of dyspepsia,—a fact which ought to have aroused my suspicion as to the different nature of the case,—and the physical examination revealed nothing but a greatly distended stomach, not dilated nor displaced. There was some nervous factor whose exact cause I did not learn, and, indeed, it is not

usually wise to probe too deeply into such matters, provided we recognize that something has occurred to disturb the tranquillity of the patient's mind, and, possibly, the innervation of viscera. Early in the evening she began to belch gas rather steadily and, for a nervous condition, rather calmly, but it is in the cases of suppressed nervousness that the most serious trouble results. The woman who cries easily and the man who swears fluently may deserve our censure for lack of self-control, but they do not usually show serious perversions of function. My treatment began with the administration of bismuth and salol, and, later, charcoal was given to fix the gas and carbolic acid as a more active gastric antiseptic. This treatment, of course, was based on the supposition that the immediate condition was one of bacterial activity with gas-formation in the stomach. After an hour of observation it became evident, first, that my therapy was not of the least benefit; secondly, that the gas eructated had not presented a sour odor at any time; thirdly, that the matter which the patient had just vomited was merely water with a little mucus and traces of medicine,—in short, that the condition was not one of fermentation in the stomach. There was no tendency to pass gas by the rectum nor was the colon distended, nor the small intestine, except, possibly, the duodenum. While a chemic investigation of the gas was out of the question, some facts can usually be readily determined, and in the present instance it was found that the gas was not inflammable,—excluding considerable percentages of hydrogen and methane; that there was no odor of sulphuretted hydrogen nor of carbon disulphide,—excluding decomposition of sulphur containing proteids by the colon bacillus, either in the intestine or, by regurgitation, in the stomach; that other odorous gases, including volatile organic acids, were absent. Now, for clinical purposes, it was well enough established that the patient was belching carbon dioxide, nitrogen,—we may include argon,—or oxygen, or a mixture of these. Meantime, I had attacked the trouble as a neurosis, using cannabis indica, atropine, acetanilide, and sodium bromide, and the belching was gradually subsiding. Let me say that, while I do not believe in polypharmacy and ready-made formulæ, emergency practice usually demands more active drugging than can be safely done with one or two substances, and I believe enthusiastically in the crossed action of medicines, giving a safe dose of several, all tending in the same direction.

For some minutes I was quite convinced that the condition was one of gastric respiration, in the mechanic sense,—that is, that the gas belched was drawn into the stomach by the suction of the diaphragm during inspiration, the cardia and oesophagus being relaxed. But more careful observation and auscultation proved that this was not the correct explanation.

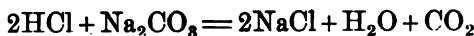
The next hypothesis was that there was a chemic respiration occurring within the stomach. At first thought, this seems plausible enough. Why should not the epithelium of the stomach, under peculiar conditions, assert the primitive respiratory function belonging to every cell and throw off appreciable amounts of carbon dioxide, just as does the epithelium of the lungs? We all know that there is such a thing as gastro-intestinal respiration, swallowed air being robbed of its oxygen, so that the intestinal gas, in the absence of digestive and putrefactive changes, consists mainly of carbon dioxide and nitrogen. Indeed, there is excellent authority for this hypothesis of gastric respiration, but a further consideration of the subject will show that it is not tenable. In the first place, while a vestige of respiratory power must be conceded to every living cell, the thick glandular membrane of the stomach is as poorly adapted to such use as can be imagined. Moreover, this hypothesis ignores the fact that respiration is not merely an elimination from the body, but an exchange, in which the body has rather the better of the bargain, taking in absolutely more than it gives off. Unless we assume a mechanic gastric respiration in such cases, we cannot claim a chemic respiration. If the former can be shown to exist, we do not need to assume the latter, while in the case in question there was no air entering the stomach through the cardia.

If we exclude mechanic and chemic gastric respiration and fermentation as causes of belching of gas, what can be the source of a volume of gas, amounting to between ten and fifty cubic centimetres, issuing from the mouth at intervals of one to five minutes, for considerable periods? Let me digress before answering this question, in order to give you the answer as it occurred to me. Some years ago I reported an approximate external method of estimating gastric acidity, depending on the production of audible effervescence when a concentrated solution of soda was swallowed. This test is quite satisfactory for clinical purposes, but it requires considerable practice to exclude the noise made by friction of garments and the ripple of gas-

tric or intestinal peristalsis, the oesophageal murmur also requiring detection, but not being difficult to distinguish. Recently, in listening to the stomach, just before applying this test, I heard a peristaltic wave in the region of the pylorus, and was surprised to hear, following this, a marked effervescence. Now, there could be but one explanation of this effervescence when no alkali had been administered: it must have been due to the mixture of the acid gastric juice with the alkaline carbonate of the bile,—pancreatic juice and intestinal juice. This production of carbon dioxide by effervescence I believe to be the explanation of the cases of continuous belching, due ultimately to a neurotic state and not explicable on other grounds. At the same time, it is evident that it would be practically impossible to collect the acid and alkaline juices separately for analysis.

This theory postulates three conditions, a sufficiency of each ingredient of the reaction to explain the belching and a state of relaxation, both of the pylorus and of the cardia. The relaxation of the cardia is undoubted, that of the pylorus is reasonable. The amount of carbon dioxide expelled at each audible belching, by actual measurement, is from ten to fifty cubic centimetres, usually from ten to fifteen. Can this amount be produced by effervescence within the body?

Physiologists agree that the bile amounts to about one thousand cubic centimetres, or one quart, and the pancreatic juice to about six hundred cubic centimetres, or seventeen ounces, in twenty-four hours; the intestinal juice has not been satisfactorily estimated. According to Gamgee, one thousand cubic centimetres, or one quart, of bile contain about ninety-five centigrammes, or about sixteen grains, of sodium carbonate, six hundred cubic centimetres, or seventeen ounces, of pancreatic juice, from twelve to twenty-five centigrammes, or two to four grains. Gastric juice amounts to about one litre, or a quart, per hour, containing two grammes, or thirty grains, of absolute gaseous hydrochloric acid. The bile and pancreatic juice may be considered as the glandular work of eight hours of the twenty-four. In the equation



the relative weights of the quantities involved, estimated by adding the atomic weights of the elements, are as follows: 2HCl , 73;

Na_2CO_3 , 108; CO_2 , 44. It is evident that the formation of hydrochloric acid is much in excess of the neutralizing power of the carbonate of sodium, and that we may pay attention only to the latter. In an hour, one-quarter to three-eighths of a grammme, or four to six grains, of Na_2CO_3 is formed by combined biliary and pancreatic secretion, hence the proportion $108:44::\frac{1}{4}—\frac{3}{8}:x$, in which x is the weight of CO_2 . This gives a value of one-tenth to one-seventh grammme, or one and a half to two grains, for x , corresponding to a volume of CO_2 of about fifty to seventy-five cubic centimetres, or twelve to fourteen drachms. Now, in secretory neuroses of the salivary glands, such as sometimes occur during pregnancy, the amount of saliva is enormously increased without a corresponding decrease of solids, and the same may safely be assumed to occur in the case of the liver and pancreas. A moderately full belching of gas, every two minutes, would require only three hundred to five hundred cubic centimetres, or ten to sixteen ounces, of carbon dioxide to be formed in an hour,—that is, only four to ten times the amount that could be formed from the normal average secretion of the liver and pancreas, disregarding the intestinal juice and the small amounts of carbonates other than sodium carbonate.

ON DIABETIC COMA AND ITS TREATMENT.

CLINICAL LECTURE DELIVERED AT THE PITIÉ HOSPITAL.

BY ALBERT ROBIN, M.D.,

Assistant Professor at the Paris Faculty of Medicine, Paris, France.

GENTLEMEN,—The subject of this lecture is one of the most serious complications of diabetes, which it will be most important for you to thoroughly understand. You all saw the patient who recently passed through our wards, succumbing rapidly to diabetic coma; his history will permit me to give a short sketch of the symptoms presented by patients suffering from this complication, of which his was a typical case.

Our patient was a railroad laborer of thirty-five, in whom we found neither syphilis nor the alcohol habit. His antecedents, hereditary and personal, were good, and he appeared to have enjoyed excellent health up to 1895, when, in April of that year, he began to notice that he was losing strength steadily, while at the same time he was tormented by intense and constant thirst, which he could only satisfy by drinking five or six quarts of liquid a day. No change in his appetite. A physician whom he then saw found polyuria and sugar, and made the diagnosis of diabetes. The man was put on a sort of diet, and continued his work, but in a month's time his strength had decreased to such an extent that he entered the hospital in June in the wards of one of my colleagues.

By that time the disease had made rapid progress. He passed nine quarts of urine per twenty-four hours, and this urine contained a large amount of sugar. Thirst had become excessive; he drank nine to ten quarts of water *per diem*, loss of flesh was very great, and his strength had totally disappeared.

He was put on a regimen and treated with antipyrin, and left the hospital on August 6, 1895, in fair condition, his urine having gone down to three or four quarts, thirst having decreased, and his strength having partially returned. He then returned to work, and we did not

hear of him again until the 17th of last March, when he was suddenly seized in the afternoon with violent pain in the left side of his chest, accompanied by very severe dyspnoea.

He remained at home six days, the physician whom he saw having applied two blisters to the left side of his chest, one in front and the other behind. Finally, on March 23, he entered our wards.

We found him lying on his back, eyes half closed, plunged in a semicomatose condition and indifferent to his surroundings. Still, he roused himself, though with difficulty, from his torpor when spoken to, but for a few moments only, lapsing into it again at once. He complained more particularly of weakness, utter prostration, to such an extent as not to be able even to move in bed, and he said that it required an enormous effort on his part to answer even in monosyllables the questions asked of him, and that all he desired was to be left alone.

His breathing was short and quick, and when we looked for the cause of this great dyspnoea we found nothing in his lungs, neither cough nor expectoration, the dyspnoea being *sine materia*. The patient complained of intercostal pain, and when near to him we noticed a peculiar odor about him, similar to that of russet apples or chloroform; the odor was that of acetone, and could be detected even at a certain distance from the patient.

The heart showed no noticeable symptoms beyond its weakness; the pulse was thin, filiform, rapid, 115 to 120 per minute. The digestive tract, liver, and spleen showed nothing either; the tongue was dry, wasted, thirst unextinguishable, and appetite wanting.

As regards the nervous system, we found the patellar reflex lacking. The pupils were normal and reacted well to light. Temperature below the normal, between 36° and 36.5° C. in the rectum. Urine relatively free, which is unusual in diabetic coma; normal in appearance, but full of sugar, very acid, and slightly albuminous. When tested with perchloride of iron, Gerhardt's reaction was very distinct. You know what this process consists in. If to the urine of an ordinary diabetic patient you add a solution of perchloride of iron and let it roll slowly down the side of the glass, the solution collects at the bottom and hardly changes color; it remains yellow and clear, and the part of the urine above it becomes cloudy, owing to precipitated phosphates. If, however, the urine belongs to a diabetic coma patient, the perchloride of iron turns very dark, dark red, like port-

wine, and this reaction is due to the presence of acetylacetic acid. Such is Gerhardt's reaction, which is absolutely characteristic of acetonæmia or diabetic coma.

Consequently, the diagnosis was certain, and the progress of the disorder was very rapid. Complete coma was present Wednesday evening, delirium during the night, and the patient died at 5 A.M.

This rapid solution in diabetic coma is the rule, and our patient reminds me of another one I had occasion to follow in private practice. By carefully following the treatment I had put him on, this man had recovered a fair amount of health. This had continued for a year, and his urine only showed traces of sugar, when he went off to do his military service on foot. He made some forced marches, and on the fourth day was seized with very violent dyspnea and was forced to return home. I found him in a state of great depression, pulse weak and filiform, temperature subnormal; forty-eight hours later he succumbed to diabetic coma.

In the presence of this extremely serious complication of diabetes, let us see what causes are capable of giving rise to it. We must be familiar with these causes, since, although diabetic coma cannot be cured, it can, at any rate, be avoided.

In the first class I place excessive physical exertion, such as is out of proportion with the resisting power of the system.

In the next class comes the absolute meat diet advised by Cantani, which decreases the sugar very rapidly, but which I do not recommend as a treatment for diabetes, as it is far from being harmless.

In the third class can be grouped all causes of nervous depression, violent emotions, and nervous disorders. In one case, a banker, who was getting along very comfortably with his diabetes, was stricken with coma after an unlucky speculation on the stock exchange which jeopardized his fortune, and died in twelve hours.

Digestive disorders form the fourth class: in ten cases of diabetic coma I found gastric hypersthenia, with hyperhydrochloric secretion, enlarged liver, and coprostasis, preceding the coma in four instances. In the other cases there was considerable organic fermentation and pronounced gastric acidity due to the presence of acetic, lactic, and butyric acids.

Abuse of the opium and belladonna treatment can also cause this coma.

In the final class can be placed such disorders as grippe, pleurisy, pneumonia, tonsillitis, etc., which also give rise to coma.

These are the causes of diabetic coma, of which the three principal clinical forms are characterized by the predominance of certain symptoms.

1. The first, called cardiac, syncopal, or with collapse, weakness, and irregularity of heart action and even asystolia.

2. The dyspnoeic form, with fearful distress and dyspnoea.

3. The nervous form, with delirium and agitation; weakness and collapse are also met with, but nervous symptoms predominate.

The evolution of diabetic coma can also be divided into three classes.

1. The very rapid form, such as that of the banker mentioned above.

2. The average form, such as that of our patient of to-day, with a prodromic period of three or four days, during which there is fatigue, dyspnoea, and digestive disorder. The patient dies in from one to four or five days, and this final drama may be caused by any slight intercurrent complaint.

3. A variety has been described with temporary or lasting remissions (provided there was not an error in diagnosis, as I have never seen diabetic coma recover).

Many hypotheses have been advanced to explain diabetic coma, but I do not intend to go into these theories in detail. For my part I have no theory. I only believe that the disorder is the result of an intoxication, the nature of which is not yet determined.

Some think that diabetic coma is due to a lesion of the nervous centres producing cerebral anaemia and oedema, disorders of the blood, and excessive accumulation of sugar; but this theory has now been abandoned. Another theory laid it to the dishydration of the blood, but experiments have shown that this does not take place. Furthermore, the inefficacy of intravenous injections of serum sets this hypothesis aside. Kidney lesions with albuminuria have also been wrongly blamed for it, as comatous uræmia, which occurs with diabetic patients, is always accompanied by symptoms that are lacking in diabetic coma.

A good deal of research has been made in this connection of late years, and has shown the presence in the system of patients succumbing to diabetic coma of various acid products, which has led Stadel-

mann to advance the opinion that the disorder is a sort of acid intoxication. I am inclined to favor this idea.

As regards the *treatment* of diabetic coma, the first question to be settled is whether there is any such treatment. For my part, I do not think there is, as I have never seen a case of this sort recover; the prognosis of diabetic coma is therefore a fatal one. Venesection has been recommended, as well as hypodermic and intravenous injections of artificial serum; but no one of these means has given any results. Lépine advises a method of treatment consisting in injecting into the patient's veins two quarts of the following solution:

R Sodium chloride, 7 (gr. xiv);
Sodium bicarbonate, 10 (gr. xx);
Aqua destillatæ, 1000 (f $\frac{3}{4}$ iv).

And in addition to this he prescribes two and a half ounces of bicarbonate of soda by the mouth. This treatment, which is based on theoretical considerations, gave an apparent result in one case, since the patient regained his faculties for a moment and asked to drink; but he relapsed into the coma and soon died.

Since, then, we cannot cure diabetic coma, it is of the highest moment to prevent it.

Any diabetic patient whose general condition begins to fail, who loses appetite and weight, is heading towards this fatal coma. To use Frerichs's expression, he is like a traveller who during a dark night strikes into a path that runs along the brink of a precipice. When, therefore, in a diabetic patient you notice the appearance of digestive and respiratory disorders, when there is a cerebral weakness or excitement, when you detect the characteristic reaction of the urine, or when you smell acetone in the breath, prescribe without delay the following treatment:

1. Stop at once all antidiabetic regimen and put the patient on a full milk diet. Avoid all untoward treatments, and be extra watchful if any intercurrent disorders appear.

2. Let out the poisons retained in the system by opening the intestinal channels of elimination by the administration of a saline purge that will not lessen the urinary secretion. I give sodium sulphate one ounce, which is at the same time a diuretic.

3. Give by the mouth each day sodium bicarbonate two-thirds ounce to saturate the acids of the system.

4. Watch the heart. If the pulse is thin, rapid, and irregular, give the following draught, which acts on the heart by its digitalis, and on the peripheral circulation by its ergotin:

R. Powdered digitalis leaves, 9 grains;
Infuse in five ounces of water, and add
Borjeau's ergotin, 1 drachm.
Dose, one tablespoonful every two hours.

If, on the other hand, the pulse is slow, soft, and without resistance, give hypodermic injections of citrate of caffeine; internally give either theobromine (forty-five grains) or citrate of caffeine.

5. To counteract the digestive disorder and to facilitate the milk diet, give, before each meal of milk, a teaspoonful of the following:

R. Strychnine sulphate, $\frac{1}{2}$ grain;
Aqua destillatae, 10 ounces;

and immediately after each meal of milk one of the following wafers:

R. Pepsin, 4 grains;
Maltine, $1\frac{1}{2}$ grains.

To prevent lactic fermentation without interfering with the action of the pepsin, give ammonium fluoride, which hinders the action of organized ferments without affecting those that are soluble. Thus:

R. Ammonium fluoride, 8 grains;
Aqua destillatae, 10 ounces.
One tablespoonful with each meal of milk.

6. Make two hypodermic injections a day of the twenty-five-percent. solution of sodium glycerophosphate.

7. Rub the surface of the body vigorously with

R. Balsam Fiosarenti,
Spirits of camphor, aa $8\frac{1}{2}$ ounces (fʒxxviii);
Tincture of cinchona,
Tincture of nux vomica, aa $\frac{1}{2}$ ounce (fʒvi. m. xl);
Essence of cloves, $\frac{1}{2}$ drachm.

8. Give the patient quantities of oxygen to breathe. This treatment is intended:

1. To keep the patient's nervous force.

VOL. III. SER. 9.—11

2. To stimulate the functions of the kidneys, intestines, lungs, and skin.
3. To alkalinize all the organs that are saturated with toxic acids.
4. To maintain the patient's strength and cardiac energy.
5. To check intestinal fermentation. And by applying it vigorously you can ward off an attack of diabetic coma that is threatening. When, however, you find yourself in the presence of a declared case, tell the relatives that nothing you can do will save the patient, as such cases do not recover.

DEATH FROM PULMONARY HEMORRHAGE.

BY FRANK C. WILSON, M.D.,

Professor of Physical Diagnosis and Diseases of the Chest in the Hospital College of Medicine, etc., Louisville, Kentucky.

THE third case that I have had in my entire personal experience of sudden death from pulmonary hemorrhage occurred yesterday. It is not often that these cases result fatally. I remember many years ago, in the Charity Hospital of New York, where I was then interne, having witnessed a death from pulmonary hemorrhage that occurred within five minutes. I had been the rounds of the ward, had seen this patient, a man suffering from tuberculosis in the third stage, with several cavities in the lung, yet in fairly good condition, and had gone up to my room on the floor above the ward in the same building, when, in a few minutes, I was hurriedly summoned by the report that this man was bleeding to death.

When I reached the ward I found the man lying across the bed with his face downward and the blood issuing from his mouth in a stream, fully as large as my thumb, and every few minutes there would be several large clots thrown out, the blood having collected in the nose and throat. It can be imagined how much blood would be lost in this way in a comparatively short space of time. The wash-basin and chamber were filled, as were other vessels that could be gathered at the moment; an immense amount of blood was lost within five minutes. I suppose it could not have been more than five minutes from the time the bleeding commenced until the man was pulseless, a corpse.

In these alarming cases of pulmonary hemorrhage, the question naturally arises, What can be done? In this case, just as in the one which occurred yesterday, the man was dead really before anything could be done. The most effective way of dealing with these cases is by the application of ice to the chest. Before a hypodermic syringe

can be gotten ready, or perhaps before ice can be applied, the patient will be dead; but the promptest way in which you can influence the hemorrhage in these cases is by pounding up some ice in a towel and applying it to the chest; the hemorrhage may be checked more promptly in this manner than by any other method that can be employed. The old-fashioned method of using salt is a very good one, and often produces a marked effect upon the circulation.

The case seen yesterday was as follows: While going to my office, about nine o'clock in the morning, I saw Mr. M., aged fifty-four years, standing on the pavement, about two doors away, talking with a friend of his. At the time he was spitting up a little blood, and this increased so rapidly that he was taken into my office as soon as possible. At the time he was almost pulseless. I gave him an hypodermic injection of nitroglycerin and strychnine immediately, as there was no hope of getting ice for local application, or even ergotin or ergotole for hypodermic use. The stream of blood from the man's mouth was now very large; the amount of blood lost was so great that it soon affected the pulse, and he was dead inside of five minutes, just as the case spoken of above occurring in the Charity Hospital, New York.

Now, I want to call attention to these unusual cases, and suggest the promptest means of dealing with them, which, as before intimated, is the local application of ice. If a physician has attended the patient before, and knows the exact location of the lesions in the lung, of course his treatment can be more intelligently applied. In the case seen yesterday, I had no opportunity of examining the chest, therefore my first effort was to ascertain the man's name, where he lived, etc., and to try and check the hemorrhage by measures that were available at the time. The man could not speak, consequently I had no means of obtaining information as to his former condition. Never having seen him before, I did not know, even, from which side the blood probably came, upon which side of the chest lesions existed. Without knowledge of this kind, the proper thing to do, where it is possible, is to fill a towel with pounded ice and apply it over both sides of the chest; if it is not known which side is at fault, apply it to both. The sudden application of cold will sometimes check the hemorrhage more quickly than anything else.

These extensive and dangerous hemorrhages may occur from the rupture of a blood-vessel of considerable size which has coursed

through tissue that has broken down, and where a cavity in the lung may have formed. In making post-mortem examinations, I have seen a considerable sized artery—a vessel, perhaps, the size of a goose-quill—running right through the middle of a cavity in the lung, and yet the vessel was intact. Nature sometimes seems to thicken and strengthen these vessels in a conservative way so as to prevent hemorrhages of this kind. I have seen instances in the dead-house where I wondered that serious hemorrhage did not occur previous to the patient's death. In other words, I have seen cavities, with twigs of blood-vessels running directly across them, entirely unsupported by any surrounding tissue, passing right into the centre of a cavity; and it is a singular thing that vessels in this way, unsupported by surrounding tissue, have not given way by internal arterial pressure, which can only be accounted for by the supposition that nature has thrown around such vessels an exudate and supportive fibrinous material, which by its organization has strengthened and thickened the walls of these vessels sufficiently to withstand the internal pressure. Where this is not accomplished, then one of these large-sized vessels may give way, slough, or burst, and these dangerous hemorrhages occur; but fortunately for these patients this seldom occurs. The case which occurred yesterday is the third that I recall in my own experience, now covering a period of over thirty years; only the third case I have met with where there was a fatal pulmonary hemorrhage. Of course, I have seen a large number of cases of pulmonary hemorrhage, but only three where a fatal issue has occurred within a very short time.

Another point to be considered in this connection is that there is very little relation between the amount of the hemorrhage and the extent of involvement of the lung tissue. In the case seen yesterday, the subsequent history shows that the man had had two hemorrhages previously, and, so far as I can learn, that was the first intimation of any trouble in his lungs. That man may have had only a very small amount of involvement of the lung tissue; and yet in his case it happened incidentally to have involved that portion of the lung tissue where the large vessels lay, and in this destructive process a break was caused in a considerable sized blood-vessel which gave rise to this large hemorrhage which proved quickly fatal. The man bled enough in a very few minutes to destroy his life. In other instances I have seen every lobe of the lung riddled with cavities from top to bottom,

and yet not a drop of blood lost from beginning to end. I have seen cases go on to a fatal termination never having lost a drop of blood; in those cases there may have been cavities which riddled the lung, perhaps more or less destroyed some of the arterial vessels or twigs; yet in all probability, in these cases, nature is supposed to block up the vessels before the sloughing process can take place, and in this way hemorrhage is guarded against. In other instances the destruction and breaking down of the lung tissue has avoided the location of the blood-vessels, which would be dangerous; the very minute twigs of the blood-vessels would be blocked up before breaking down of the tissue would occur, and in this way hemorrhage has been guarded against. But I want to emphasize the importance of not drawing any inferences as to the amount of lung tissue involved simply from the occurrence of hemorrhage, no matter what may be its extent; for a large amount of lung tissue may be involved in the tuberculous process and yet not a drop of blood will be lost; again, a patient may have alarming hemorrhages, one after another, with the smallest amount of lung tissue involved, so small an amount, in fact, that frequently the physician is not able to detect it by means of delicate appreciation of the physical signs,—that is, the tuberculous deposit may be so slight as to be hardly detected by a careful and minute physical examination.

It is well to review these emergency cases, as any one may be called to such patients in practice, and also to point out the measures to which the attendant may resort in an effort to benefit the conditions present. In the first place, the application of cold to the chest; second, the hypodermic injection of ergotin or ergotole,—the latter being a pure fluid extract, but may be used hypodermically; third, the hypodermic use of full doses of an opiate (morphine) which serves to quiet the circulation, and in this way lessen the tendency to hemorrhage; fourth, absolute quiet on the part of the patient in the semi-recumbent posture. If the patient is kept quiet, of course that assists in quieting the circulation; and then an effort on the part of the patient to restrain the act of coughing will also assist in lessening the tendency to hemorrhage,—in this way tiding over the immediate danger from hemorrhage, and the patient will in all probability soon recover his strength, his tone, and the attack may in this way be tided over. If the hemorrhage can be checked for twenty-four or thirty-six hours, usually nature will succeed in

blocking up any break in the blood-vessels which may have occurred, and the danger will have been passed. Do not infer, from the spitting up of dark clotted blood the next day, that a fresh hemorrhage has taken place; for, of course, there will likely be some remnants of clotted blood left in the bronchial chambers which may be brought up by the effort of coughing the next day or the day following; and if it is dark and grumous in its character, it does not indicate a fresh break in the blood-vessels, but simply remnants of clots that have been retained in the air-passages and which are loosened in the act of coughing and expectorated, just as any foreign substance would be brought out of the respiratory passages. If the hemorrhage is bright red, then, of course, it would indicate a fresh break in the blood-vessels, which would call for prompt action to prevent its continuance.

There is another source of hemorrhage which may sometimes be met with, and I have seen two cases of this kind, where it may be difficult to determine whether the blood comes from the pulmonary tissue, or whether it comes from some other locality. We occasionally have hemorrhage occurring from the throat. In those cases the blood will be less mixed with air, more like ordinary blood, and not so frothy as that which comes from the deeper lung tissue. If blood comes from the lungs, of course the air circulating in and out through it will necessarily mix it more or less with air-bubbles, which would give it a frothy character. On the other hand, blood that comes from the throat is less apt to be mixed in this way with air than the blood that comes from the pulmonary tissue.

Then there is another source of hemorrhage, and, as above stated, I have met with two instances of the kind, where the blood comes from the rupture of an aneurism in the immediate neighborhood of the trachea or the large bronchi, an aneurism, say, of the descending arch of the aorta. Under these circumstances the physician may be called upon to see a patient, hurriedly called in an emergency, because of sudden and alarming hemorrhage; he may know nothing of the history of the case, and it would be well to recall the points of differentiation between hemorrhage from pulmonary tissue and that resulting from rupture of an aneurism in this situation, because under those circumstances examination of the case will have to be made very hurriedly. If he has attended the case before, most likely a condition of this kind (aneurism) would have been detected previ-

ously. For instance, an aneurism involving the descending arch of the aorta would in all probability give rise to certain symptoms which would make it very apparent. Now, the occurrence of a pulsating tumor, probably resting upon the spinal column, will produce an erosion of the vertebral bones themselves by the constant pulsation. A tumor like an aneurism in the immediate neighborhood will actually necrose and break down the integrity of the bodies of the vertebrae, and will produce the peculiar character of pain of which these patients complain. They will state that they feel as if some one was boring with a sharp instrument into the spinal column itself. They will describe a peculiar boring, gnawing pain, which is produced in this way by constant breaking down of the bodies of the vertebrae; sometimes one, two, or three of the bodies of the vertebrae may be involved, and there may be actual breaking down of the bone, and in this way sometimes the development of kyphosis, actual curvature of the spine, the breaking down of these bodies allowing the spine to descend from above, resulting in a decided deformity; but I call attention especially to the peculiar character of pain of which these patients will complain. When an examination of such a patient is made some prominence of the spinous processes may be felt at that point; there may be a considerable degree of tenderness over that area, and an aneurismal bruit to which the condition gives rise may be detected. Where the physician has an opportunity to examine the case previously, of course he has detected this condition, and the possibility of rupture of the aneurism into the trachea or into the main bronchus would occur to him, and would be thought of at once as the possible source of hemorrhage. Again, an aneurism affecting a large vessel, if it is in the immediate neighborhood or close to the arch of the aorta, would in all probability produce some laryngeal symptoms from pressure upon the recurrent laryngeal nerve. Occasionally we can diagnose an aneurism of the arch of the aorta, or of the first portion of the descending thoracic aorta, by noticing paralysis of the vocal cords, attributable to implication, pressure, and paralysis of the recurrent laryngeal nerve. Where this has been diagnosed, if a large fatal hemorrhage occurs, then, of course, it would at once be concluded that the hemorrhage was from that source, particularly if the patient gave no history of any pulmonary trouble previously. In a case of this kind little or nothing can be done towards relief; a fatal issue is almost inevita-

ble, and the only thing left is to render what assistance we can to make the patient more comfortable while he lives, because nothing can be done in the way of relief by medication.

In cases of pulmonary hemorrhage, excepting in those cases where a fatal amount of blood is lost in a few minutes, such as I have described, the immediate application of ice, the hypodermic injection of ergotole, etc., will, as a rule, be followed by a cessation of the hemorrhage, and thus a fatality may be avoided.

RUBEOLA; TYPHOID IN CHILDREN; SARCOMA OF THE LIVER.¹

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF JENA.

BY PROFESSOR LUDWIG KREHL,

Director of the Polyclinic and Professor of Special Pathology and Therapy at the
University.

GENTLEMEN,—I have some cases of an eruptive contagious disease in children to show you this morning that are interesting because they are specimens of a disease whose independence is not even yet generally acknowledged by all physicians. The disease possesses its only importance from a diagnostic stand-point; its course is invariably mild, its treatment the simplest and most natural hygienic precautions; complications do not occur; sequelæ it has none. Its close resemblance to measles in most cases, and a certain similarity with scarlet fever in other cases, make it important that the disease should be carefully differentiated from these two at times serious and under all circumstances never-to-be-neglected affections. It does not confer, despite the similarity of the eruption in many cases, any immunity for either of these two diseases, so that it is highly important to know for certain in any given case whether a child has had the milder rubeola, or measles, or scarlet fever, for its proper protection from exposure to contagion in future epidemics will depend upon exact knowledge in this regard.

We are just in the midst of an epidemic of fever, accompanied by an exanthematic rash, among the children of Jena, which I thought at first was a very mild form of measles. The rash has many of the characteristics of that disease; there is slight fever; there is even some tendency to snuffles and conjunctivitis,—the snuffles so characteristic of measles. I have within the last few days seen a

¹ Reported by James J. Walsh, Ph.D., M.D.

number of cases, however, where the children had already had measles, where, in fact, I had myself had them under my care as measles patients, and where I cannot have any doubt about the diagnosis. In Wenigen-Jena, where the present cases occur, unhygienic surroundings, or, rather, the lack of proper sanitary precautions, greatly facilitate the spread of contagion, so that, thanks to this, I have now seen a number of cases of the disease where I before saw measles.

While a second attack of measles is not out of the question, and undoubtedly occasionally occurs, still it does not occur frequently. It is a medical rarity, almost deserving special notice, not the same thing that we might reasonably expect to find in a series of cases as here. There is no doubt to my mind, then, that we have to do with that other exanthem, rubeola. The undecided stand of certain of the great German paediatricians—notably Henoch—as to the absolute independence of the disease from an etiological stand-point, and the possibility, not yet entirely excluded, of the disease being a degenerate form of measles, has given rise to a good deal of discussion as to the true status of the affection even here in Germany, the supposed home of the disease, if we may trust the name so commonly given it by other nations, German measles.

While we do not know the cause of measles, and *a fortiori* not of rubeola, we cannot absolutely say that the one may not have certain etiological relations with the other, but to any one who has seen clinically an epidemic of an exanthematous febrile condition, in which case after case occurred where the child had previously had measles,—measles not merely diagnosed by the mother or some passing neighborly wiseacre, but by a physician on whom absolute dependence could be placed as to diagnosis,—and where even the records of the previous cases are at hand to substantiate the remembrance, there can be no doubt that there is an exanthem independent of measles at work.

The two cases we have here this morning have a certain similarity in themselves, and approach, as you will see, to a certain extent the scarlet fever type rather than the measles type in the appearance of the eruption. There are a number of small, red, almost punctate spots, slightly raised, in places grouped so close together as to simulate the diffuse redness of scarlet fever. This on the face and neck, while the spots are larger with a cast of blue in them here on the arms; this

is much more like measles. In scarlet fever the chin is always free from the eruption, which is not the case here. There is a profuse redness of the tonsils and pharynx here, but no deposit on them, as is usual in scarlet fever. Then we have those characteristic signs of measles so rare in scarlet fever,—slight conjunctivitis and bronchitis.

Though we have this eruption that you see practically all over the body, there has never been any fever, and the eruption has all come out in the last twenty-four hours. In scarlet fever at this stage we would surely have febrile temperature, and in measles the temperature would have at least preceded the exanthem. With the children undressed you see other characteristics of the eruption that distinguish it from scarlet fever. The exanthem, though composed of distinct little red spots here on the body, does not show that tendency to confluence with the production of diffuse red patches that is so characteristic of scarlet fever. Besides, the inguinal and axillary folds are not reddened here as they almost invariably are in scarlet fever.

We have, then, to do with a febrile eruptive disease, that is related to measles and scarlet fever, yet is much milder than either, and possesses certain characteristics that differentiate it from both. The two cases we have here have definite similarities in the eruption, and in a given epidemic this seems to be generally the case. If we compare the notes of different epidemics even, as described by careful observers, the similarity vanishes. For some this type of disease rötheln has the diffuse punctate scarlet eruption we have here, for others the patches are larger in area, resembling measles very closely; and then a series of cases occur with gradations in the exanthem between measles and scarlet fever.

It is very possible, then, that we have, under the name rötheln or rubeola, a number of slight infections not necessarily due to the same cause. The one characteristic of the disease that all observers are agreed on is its extreme mildness. Until we know the exact etiology of the exanthematous diseases, and are also able to study the specific biology and morphology of their microbes, we cannot say definitely. There may be, of course, and probably is a whole series of microbes capable of producing this mild exanthem and a number of them will without doubt be distinguished before long. Meantime, rubeola makes a name for these cases that is as good as any other, and

whose use emphasizes the fact that no protection is to be expected after an attack of it from either measles or scarlet fever, and this is, after all, the important point in the matter.

The treatment is, of course, of the simplest description. The children should be kept in bed for some days, on a mild, mostly liquid diet, though, as they usually have a good appetite, I think it an unnecessary discomfort to limit to any great degree the amount of food they may wish to take. After four or five days the eruption disappears, and after two weeks I think the children may be allowed to go to school again. There is no legal regulation in the matter, and this time I consider long enough.

Our next patient is this little child, whom you can see is very ill. It is about a year old, and had during last summer a severe attack of summer diarrhoea. Its general nutrition suffered a good deal at that time, and it never entirely recovered. This is partly the reason for its miserable appearance now, though there is another excellent reason in its present illness. I may remark in passing that there is no luetic hereditary in the case, so that none of its poor condition is to be attributed to that. I mention it because some of the worst cachectic conditions in children are of course luetic, and the young physician is apt to think of it in every case.

The child has been ill now for some time. The temperature this morning is 40.3° C. (104.5° F.), a very high febrile temperature. The mother says the child has had no fever for five days before yesterday, but that for two weeks before that it had had high fever every day. Examination shows a tensely distended tympanitic abdomen, meteorism, in which, despite the increased tympany, we are able to demonstrate an enlarged spleen. Besides this, there is a persistent obstinate bronchitis.

With a temperature as high as this we look for all possible sources of infection; examine the ear and the nose and the mouth, to be sure there is no collection of pus from which toxines are being absorbed; examine the child all over for a concealed abscess. We think of tuberculosis, of course, but there are none of the signs of meningitis, only bronchitis, though very persistent, in the lungs, and meteorism, not ascites, in the abdomen. Cold abscesses usually give no such acute symptoms until after being opened, and, besides, there is no sign of any such chronic localized process.

That we have to do with an infection, however, seems clear. With

the meteorism, the enlarged spleen, and the bronchitis, we can only think of typhoid fever. The continuous fever for two weeks, which was treated as an intestinal catarrh, was probably typhoid, and here we have to do with a relapse. Typhoid is rare in children of a year old, yet not so rare but that we must think of it under circumstances like these. It is just possible that this is still merely an autointoxication from the absorption of toxic by-products in a diseased intestine, but that would not explain the bronchitis which is so common, on the other hand, in typhoid. The child comes from the suburb of Wenigen-Jena, where, owing to the absence of proper water-supply and a sewage system, typhoid is epidemic.

The treatment will of course be merely symptomatic; the child must have cold baths when its temperature rises above 103° F., and absolutely nothing to eat except milk. The question of prophylaxis here for the rest of the children—there are six of them—is extremely important. So far the child has been taken care of by its nine-year-old sister during the day, the mother having to go out to work to help support the family. This state of affairs must not be permitted to continue any longer.

When children are ill with typhoid the danger of dissemination of the germ to other members of the family is much greater than with grown people. Their stools are received in cloths that are handled and washed, and the child itself is apt to be caressed by other children in the family. We cannot trust the taking of precautions against infection to a child of nine, so I shall have to state very clearly to the mother the danger to her other children, and insist on her remaining at home to take care of the child. The diapers will have to be immersed at once in a strong solution of lime, every precaution of cleanliness will have to be taken, and the other little ones will have to be kept away from the sick child as much as possible.

Our next patient, gentlemen, is a man of forty-five, who comes complaining of stomach symptoms. About eight weeks ago he lost his appetite,—not that he had nausea or vomiting, but simply he did not care to eat. He forced himself to eat really more than he cared to, but in the meantime he has noticed that he has grown weak and thin. He has never had any stomach symptoms before. He has always had an excellent appetite, and his bowels have been perfectly regular. He has not abused alcohol, and has practically never before been sick in his life. This point he insists on very much.

This was all the history he gave us until, noticing the absence of his left eye, we asked him how he had lost it. It was removed two years ago by Rindfleisch, of Weimar, he told us, because of a tumor that was growing in it. We were not so surprised then to find ourselves, on examination of his abdomen, in the presence of a tumor of the liver. There is a general enlargement of the liver that causes its lower edge to descend almost to the umbilicus. There are, besides, a number of prominences to be felt on its surface, especially one here slightly to the right of the median line. We have evidently to do with a neoplasm, or rather series of neoplasms, of the liver.

Inquiry shows that the ocular tumor removed was a melanosarcoma of the retina, and it is well known that these often give metastases to the liver. Naturally one would expect that a tumor of this size, and causing the loss of appetite noted in the case, very probably because it interferes with the function of so much of the liver substance, would have produced more cachexia than this. It is typical of sarcomata of the liver, however, that they run a course that is often almost symptomless until very near the fatal termination. Still another characteristic of these melanosarcomatous metastases from the eye is that they often occur a good while after the removal of the original tumor. They have been known to occur as long as ten years after the enucleation of the eye, so that in these cases it is nonsense to talk of a radical cure after operation until almost a score of years have passed. In some of these cases melanin has been found in the urine of patients with hepatic melanosarcomata; here so far we have not found it.

The treatment of the case can of course be only symptomatic. Operation is out of the question. We may be able to restore his appetite, in part, at least, by tonic appetizers,—by hydrochloric acid and some of the stomachics. We must, however, take every precaution that only the most unirritating food shall be taken. His liver, so important for intestinal digestion, has practically lost its function in the growth of the neoplasm amid its cells. He will have to take mainly a milk diet, to which a certain number of soft-boiled eggs will be added each day, according as his general condition seems to call for nutrition and his appetite permits the reasonable expectation of their digestion without dyspeptic symptoms. Fats he will have to avoid as a rule entirely, because their absorption depends so much on the biliary secretion. Even milk he will be advised to take de-

prived of its cream. Starches he must take only with the greatest moderation.

The condition of the bowels will have to be watched with the greatest care, and no accumulation of faeces permitted. With the biliary secretion lessened by the presence of the neoplastic elements in the liver, the antiseptic influence of the bile is lacking in the intestine, so that accumulation of faeces would be the signal for decomposition of intestinal contents, and would be rapidly followed by the absorption of toxic products and symptoms of autointoxication. This must be avoided by the use of gentle purgatives when needed, and the occasional use of calomel in divided doses, which at once stimulates liver secretion, acts as an intestinal antiseptic, if any drug really fulfils that function, and produces gentle catharsis. As painful symptoms develop, morphine will have to be freely used, and euthanasia secured as far as possible.

THE BEARING OF EMBOLISM ON THE COURSE OF VALVULAR HEART-DISEASE.

BY WILLIAM M. GIBSON, M.D.,

Utica, New York.

IN giving the prognosis in valvular lesions of the heart or in aiding the patient to plan for his future there is probably no more disturbing element to be considered than the occurrence of an embolism. If in these cases there exists proper compensation for the obstructing lesion or insufficient valve, we generally expect a reasonably good length of time to elapse before the compensation begins to fail; provided, of course, no reinfection of the lesion occurs and the patient is not subjected to mental or physical strain, or weakened by illness; and when the compensation begins to fail due warning is generally manifested either in dyspnoea or interference with digestive or renal functions, or by the commencement of oedema in some part of the body.

Gibson, in his "Diseases of the Heart and Aorta," in considering the prognosis and treatment of chronic affections of the heart, writes as follows: "From the length of time which has elapsed since the commencement of any valvular lesion it is possible to predicate to some extent what will be the probable future of the lesion, for in cases which have existed for a considerable time without serious inconvenience there will naturally be a hopeful expectation. When a tendency to dilatation disturbs the compensatory process of hypertrophy the prognosis is serious, and any tendency to cardiac failure or asystole is of evil omen. . . . The effects of the lesion upon the system elsewhere must be judged by the condition of the digestion, of the blood, of the lungs, of the kidneys, and of the brain. . . . When attempting to make a forecast of the future and to lay down rules for the guidance of any case of valvular disease the age and sex of the patient will necessarily set natural limits to the possibilities of management; it is clear that the conditions are subject to entirely different agencies when the valvular disease occurs in early, middle, or

later life, and in almost the same way the sex of the patient exercises a preponderating influence."

It is evident that these conclusions are based on a study of the part played in valvular heart disease by compensatory hypertrophy. An embolism, however, often occurs when compensation is at its best and when the person is not prevented from attending to the matters of every-day life.

Gerhardt's advice, not to over-seriously consider a murmur which exists in a heart not increased to the right and in which the apex beat is not out of normal position, or the second pulmonary sound is not accentuated, is probably a suggestion we all accept; but a few vegetations on the valves of the heart may give rise to a murmur and yet offer no great obstruction to the blood current. So slight an obstruction will hardly call for increase of heart-muscle enough to displace the apex or extend cardiac dulness to the right. The detachment of one of these vegetations may produce fatal cerebral embolism or give rise to serious disturbance of digestion or kidney function, or produce gangrene in an extremity. The age, sex, and environment, which, as Gibson says, necessarily limits the management of valvular heart-disease, give us no intimation of the danger of the occurrence of an embolism.

Emboli swept into the circulation arise chiefly from vegetations formed on the valves of the heart, from atheromatous changes in the aortic valve, and also from coagula formed in the heart cavities and pulmonary veins. Thrombi in the veins of the lower extremities furnish embolic masses which are frequently the cause of hemorrhagic infarction of the lungs. The consequences of an embolism depend very much on the nature of the embolus, the area of the blood-vessels occluded, and the function of the organ disturbed (Weichselbaum). The effect of an embolism on heart-action is modified somewhat by these same considerations; but the nature of the infarction, whether it is anæmic, hemorrhagic, or septic, must also be taken into consideration. Recently formed coagula or vegetations causing infarction will be absorbed more quickly than older ones; and probably emboli thrown off from the valves soon after an acute endocarditis carry the infecting germ, although the febrile symptoms may be in abeyance. Infarctions which are anæmic will disturb compensatory heart-muscle less than hemorrhagic infarctions, unless large areas or vital organs are involved.

An embolus may travel wherever the blood-current offers a channel for it, but its direction seems to be special rather than general, the ramifications of the pulmonary artery being most frequently affected. The vessels of the lower lobe of the right lung are the most often occluded; the power of the blood-current and the weight of the embolus are (according to Eichorst) responsible for this selection. The renal, splenic, mesenteric, and cerebral arteries and the branches of the celiac axis appear to suffer next in frequency. The condition of the heart wall, while it may not be especially concerned in the detachment of masses from the valves, has much to do with the formation of coagula. Mitral disease, causing considerable hypertrophy and dilatation of the right cavities, is frequently complicated by pulmonary embolism.

Recurring pulmonary hemorrhages in mitral disease are generally the results of infarctions. Slight hemorrhages often relieve an over-worked heart and engorged lungs, but the more profuse bleedings exercise a baneful influence on heart-muscle. The loss of blood is often difficult to make up, and the long confinement in-doors necessitated deprives the patient of the much-needed open-air life. The pneumonias following infarctions also tend to destroy compensatory muscle and interfere very seriously with general nutrition.

A cerebral embolism may give rise to disturbances of brain function, ranging anywhere from slight disturbance of consciousness to a fatal apoplexy. Infarction of the renal vessels remaining purely anaemic will probably not disturb the heart's action very seriously, but suppurating areas developing afterwards may give rise to very grave conditions and ultimately renew endocardial inflammation. Infarctions of the branches of the celiac axis are frequent, but escape notice in many instances. In the mesenteric arteries they may give rise to serious peritoneal inflammations, and even gangrene of an intestine.

Infarctions of the peritoneal blood-vessels during the course of acute endocarditis, or after the subsidence of the acute symptoms, may give rise to symptoms demanding operative relief. Such complications should be carefully studied, and the assistance of an experienced surgeon will often aid in detecting septic processes which may escape the notice of the general practitioner.

A little over a year ago a young married woman was brought to one of our hospitals, apparently in a serious condition, owing to some obscure abdominal infection. Shortly before the appearance of the

abdominal symptoms she had passed through a rheumatic endocarditis, rather severe in type and of considerable duration. An exploratory laparotomy failed to discover any disorder of the uterus and its appendages or the appendix. Her symptoms, however, persisted, and after a few weeks became so serious that another operation was decided upon. A distinct tumor could now be made out in the region of the right kidney. At the second operation it became necessary to remove a large amount of inflammatory tissue and adhesions which had occluded the right ureter. The kidney was found a cystic mass, which was also removed, and the woman then made a good recovery. It was, of course, impossible to detect the site of the original infection, but it seems more than probable that the infecting material had been conveyed by embolism from the endocardial inflammation.

Pepper, in his article on "Diseases of the Endocardium," in the "American Text-book of the Practice of Medicine," calls attention to the occurrence of small infarctions in the vessels of the skin, producing petechial eruptions and also areas of subdermal effusion. A case of mitral regurgitation with good compensation, under my observation, developed two of these areas, one in the skin of the left thigh and another on the outer surface of the right leg above the ankle, which remained for weeks in a low state of nutrition bordering on gangrene. The effect on compensation was very marked, and the patient has not succeeded in regaining the loss due to this long drain on nutrition.

Gangrene of the extremities following embolism is necessarily of very grave import in valvular heart-disease. The tax suddenly thrown on compensation by the occurrence of gangrene is liable to produce extreme dilatation, even if the patient rallies against the general effects of septicæmia.

During the last epidemic of influenza I saw, with Dr. A. R. Simmons, of Utica, a case of mitral stenosis of long standing that had suddenly developed gangrene of the right foot and leg. Influenza with a right lobar pneumonia had preceded the gangrene, but the compensatory hypertrophy had not suffered very much. Amputation above the knee arrested the gangrene, and the stump healed kindly. A recurrence of pneumonia, probably embolic, proved too much for the patient's strength. An examination of the amputated leg disclosed an organized clot, nearly two inches long, at the bifurcation of the popliteal artery. The probabilities are that with good care this

woman would have again secured a fairly good compensation, even after the long strain due to influenza and pneumonia, had not the two infarctions occurred.

The following cases are reported as illustrative of the effects of embolism in heart lesions which were not judged to be of a serious nature:

CASE I.—A gentleman, aged thirty-nine years, whose family physician had discovered a heart murmur when looking him over for some minor affection, came to me for further advice. While there was an unmistakable murmur at the aortic valve, I could not make out that cardiac dulness was increased to the right or that the apex beat was out of normal position. Apparently the lesion—aortic obstruction—was of slight concern, for he never complained of dyspnoea, and the renal and digestive functions were perfectly carried on. While sitting in his office, perhaps half a year after I saw him first, he was taken suddenly with giddiness, vomiting, and partial aphasia. There were no symptoms of paralysis in the extremities, but it was fully three weeks before he recovered full use of the speech centre. Two years later he was taken in a similar manner, and suffered from converging strabismus of the left eye; there was also considerable mental depression accompanying this attack. During the next few years he had six attacks, all of which were accompanied by vomiting and some impairment of intellect. In December, 1896, while talking with his family, he was seized with a right hemiplegia and aphasia. By the end of the following March he had regained muscular control and the power of speech, and was apparently as well as ever. A month later he passed suddenly into coma, which terminated fatally in two days.

CASE II.—A woman, aged thirty-six years, during her second pregnancy, suffered an attack of acute rheumatism in the second month, which lasted about two weeks. She had a recurrence of the rheumatism in the fifth month, which caused a slight endocarditis, but from that time on was free from rheumatism or any febrile disturbance. She carried from a former attack in childhood a heart-murmur which was heard with the first sound of the heart over the aortic valve. She had been carefully examined by several physicians, who pronounced the murmur to be of no serious consideration. The only symptom referable to a disturbed heart that she had ever complained of was a slight but persistent dyspnoea that occurred when travelling or living

in elevated regions. I had examined her heart from time to time during a period of three years prior to her pregnancy, and while the murmur was always present, no increase of cardiac dulness could be detected. There was, however, slight accentuation of the second sound. She had a normal labor and convalescence up to the eleventh day. On the evening of that day she was taken with a severe chill, which was repeated in four hours. These rigors were followed by a rise of temperature to 105° F. and profuse sweating. The chill recurred at noon the next day. A careful examination of the uterus and appendages was made by Dr. J. H. Glass, who pronounced them free from infection. Nothing abnormal could be discovered in the lungs. The examination of the urine, however, showed it to be highly albuminous, and numerous granular casts, studded with pus-corpuscles, were found under the microscope, but there were no evidences of cystitis present. Urinary examinations had been made several times before parturition, and also afterward, but nothing abnormal had been detected prior to the chill. Two days after the chill the patient complained of pain and soreness in the region of the left kidney. The urine became scanty and high colored and the amount of albumen increased; the pus casts became more numerous, and the patient passed into a serious condition of septicæmia, which lasted about two months, but finally made a good recovery.

I am very certain in this case that there was no external cause for the suppurative nephritis, and have always believed that it was due to an embolism of a branch of the renal artery, and that the infection retained from her previous endocarditis changed the infarction into an area of suppuration. This woman has borne three children since without a recurrence of any renal disturbance, and the condition of the heart wall remains practically unchanged in spite of the septicæmia; but in her case the question of the recurrence of an embolism may be a more serious factor in the next few years than endocardial change and slow impairment of heart-muscle.

CASE III.—Last fall a gentleman, aged seventy years, was thrown from his bicycle and sustained a fracture of the left humerus. He had suffered several attacks of acute rheumatism, scattered over a period of thirty years, but had escaped endocardial disease until the winter previous to his injury. An attack of rheumatism in January was complicated by endocarditis, which was aggravated by another attack in March. He was soon able, however, to take his place in his

bank with very little inconvenience. The month of June he passed at the sea-shore, where he regained his usual strength. An examination of his heart at this time disclosed slight murmurs at the aortic and mitral orifices; the cardiac dulness was not much increased, and the position of the apex beat was nearly normal. An examination again in September showed about the same state of affairs. Aside from slight dyspnoea, which came on from riding his wheel up steep grades, the man was apparently free from symptoms of cardiac disease. Immediately after his injury he was placed in bed, and after the shock consequent to the injury had passed off the fracture was reduced and bandages were applied. He remained in a very comfortable condition until the next afternoon, when he was suddenly taken with a sharp, stabbing pain under the umbilicus. The pain became more and more violent, and demanded the free use of morphine for its relief. The day following the pain was even more intense, and was not relieved by a high enema which brought away a large amount of faeces. The temperature remained normal, but the pulse was hard and somewhat accelerated. Symptoms of intestinal obstruction now developed, and the condition of the patient became very grave. His wife and son and the patient himself begged for operative interference. With the hope that possibly an old hernia might in some way be the cause of the intestinal obstruction, an exploratory laparotomy was performed by Dr. Kilbourne and Dr. J. D. Jones. As the intestines were laid out on the sterilized towels a gangrenous fold of intestine rolled into view. It was found that the supplying mesenteric artery had been occluded by an embolic mass. The intestines were drained and the abdominal incision closed, as it was evident that the gangrenous portion of the intestine was too large to permit of its removal. The patient died of exhaustion the day following.

The occurrence of embolism during the course of valvular heart-disease is certainly not an infrequent complication, but prophylaxis offers little or nothing for its prevention compared with the effect of good management on compensatory hypertrophy. Recognition of even a slight embolism, however, is of great importance, for much can be done through rest and administration of proper remedies, surgical relief included, towards preventing some of its more serious results.

THE COMPLICATIONS OF CROUPOUS PNEUMONIA.

BY JOHN G. CECIL, B.S., M.D.,

Professor of the Principles and Practice of Medicine and of Clinical Medicine in the Louisville Medical College, etc., Louisville, Kentucky.

PLEURISY is the most common complication of pneumonia, and the fact is that inflammation of the pleura is associated with nearly every case of pneumonia. It is only where the disease, as an inflammatory process, is limited to the central portions of the lung, that the inflammation does not extend to the surface. In every case where it does extend to the surface there is a pleurisy. This may be so simple as to only involve a small spot of the pleura, giving rise to a local point of tenderness or pain, with no disturbance other than that, or it may amount to an extensive involvement of the pleura with all the train of symptoms and results incident to such involvement.

Next to pleurisy pericardial and endocardial troubles are common and important complications. In certain forms of pneumonia, especially in the malignant types, there often appears an endocarditis or pericarditis, either of which becomes a dangerous complication. The principal danger in pneumonia is from the heart, and when to that is added a suppurative endocarditis or a pericarditis, naturally the case becomes more grave.

Pericarditis is extremely hard to differentiate from pleurisy, especially if it occurs at the base of the left lung where the two membranes are in such close contact. It may be impossible at times to make the differentiation. Usually, however, in pericarditis there is more or less effusion into the pericardium, and an irregular action of the heart, which is not the case in pleurisy. The well-known physical signs of pleurisy may be distinguished, but possibly not those of pericarditis. In pleurisy there will be detected the pleuritic friction râle or rub with each act of respiration; in pericarditis the friction râle will be heard with each action of the heart. One can differentiate between the two at times, when the râle or rub is plain, by ask-

ing the patient to cease breathing for a moment. Of course, as he ceases breathing, the pleuritic râle will cease. If he ceases breathing and the râle continues, and is detected with each action of the heart, it becomes plain that it is a pericardial friction râle. This is one of the best means of differentiation. If there is an effusion with the pleurisy, if the pleural cavity is partially filled with fluid, the physical signs of this effusion will be evident; the lower part of the pleural cavity will be filled with a line of dulness entirely around the lung. In pericarditis with effusion there is an increased area of dulness over the heart, that far-away sound of the heart at its apex, because the apex of the heart is pushed away from the chest wall by the effusion; an irregular action of the heart with signs of dyspnoea, which would not be as easily accounted for as by the pneumonic process.

In pericardial effusion the area of heart dulness is much enlarged and the base of the triangle is reversed. In percussing the normal heart the base of the triangle of dulness is up, corresponding to the base of the heart, the apex being below, corresponding to the apex of the heart. In pericardial effusion the base of the triangle is reversed, the area of dulness being greater at the apex of the heart than at its base. It is a valuable aid if this increased and inverted area of dulness caused by effusion into the pericardial sac can be mapped out.

If the patient lives long enough after development of endocarditis, there is evidence of it in pain, depressed action of the heart, sometimes an intermittent action; the heart is apparently very sensitive to all impressions, acts in a very irregular way, sometimes fast, then slow (generally fast), irregular, and weak, with other evidences of sepsis, because suppurative endocarditis means that there is suppuration in the interior of the heart, and when this is true it is not long before evidences of septic trouble become manifest throughout the system. This would be shown in the development of abscesses in different parts of the body, infarctions in the lung, evidences of meningeal inflammation, apoplexy, paralysis, and such evidence as would be shown by the carrying and depositing of septic and inflammatory products by means of the blood to any organ of the body.

Meningitis is another complication which is not uncommon, and is shown by a group of symptoms which are sometimes obscure, sometimes plain. In ordinary cases of meningitis complicating pneumonia from the start there will probably be increased nervous phenomena, great restlessness, rolling from side to side, sometimes opisthotonus,

generally a mild delirium or a mania; sometimes the delirium is quiet, but in all cases delirium is a prominent symptom. With meningitis as a complication there is also frequently an irregular state of the pupils,—one contracted and one dilated; paralysis or evidence of central involvement may be manifested in twitchings of the muscles here and there.

Relapses in pneumonia are uncommon; in this respect it is directly opposite to typhoid fever and some of the other fevers. In typhoid fever relapses are common, recurrences rare; in pneumonia relapses are uncommon, recurrences are very common. A man who has had pneumonia is always liable to have it again. Many people will live through a number of attacks of the disease; it sometimes comes on after very slight exposure year after year. One instance is on record where a man had twenty-eight attacks of pneumonia,—a case recorded by Bush.

Of the clinical varieties of pneumonia, we have several. As would naturally be expected, there is a malignant form of this disease just as there is of all other specific diseases. We have malignant scarlet fever, malignant small-pox, malignant measles, malignant malaria, etc.; we have also malignant pneumonia, in which the impression is very profound from the beginning. The patient may die before many of the characteristic symptoms have developed, even so quickly as to make the diagnosis doubtful. In many cases of the malignant type patients will succumb inside of three days, several days before the crisis might reasonably be expected. Again there is a malarial type of pneumonia, occurring in connection with malarial fever, which is not unusual except that it is recognized as a form of pneumonia which is associated with malaria and is more common in malarial regions than elsewhere. It is a complication which should be borne in mind, as when these two diseases coexist the prognosis is correspondingly graver. In the adynamic type of pneumonia there is a profound impression upon the heart from the beginning, extreme weakness, perspiration, and a generally bad outlook. Persons with Bright's disease or diabetes, or with tubercular lung trouble, if they develop pneumonia, are likely to have the adynamic or asthenic type. There is still another form which occurs frequently, known as typhoid pneumonia. This, however, has nothing whatsoever to do with typhoid fever. Typhoid fever may be complicated by lobar pneumonia, but this is not typhoid pneumonia. Patients with pneu-

monia sometimes get into that state known as the "typhoid condition." There is a low muttering delirium, a brown, furred, dry tongue, weak pulse, and all the other evidences of the so-called typhoid condition. There is not a separate germ which causes the disease called typhoid pneumonia, and it may occur in any case of pneumonia in which the progress is bad. The epidemic form of pneumonia which prevails at certain periods is more dangerous and seems to be more fatal than any other form of the disease. Certainly it is when we find it accompanied by influenza, breaking out in institutions, schools, etc., such as has been seen in Louisville during the prevalence of la grippe; but those cases I have seen within the last year have been of a rather mild type.

In chronic alcoholics—drunkards—the prognosis of pneumonia is always extremely grave. The disease may be overlooked in that class of patients, or may not be recognized until the presence of prune-juice sputum is detected. A man with delirium tremens has to be watched very closely; at times he will develop pneumonia without any distinctive symptoms; the sputum will not be characteristic, and the impression made upon him by the delirium tremens may be such as to cause the development of pneumonia to be overlooked.

Croupous pneumonia develops in children oftener than we have been led to believe. It is not a common disease in very young infants, not nearly so common as broncho-pneumonia, nor is it so dangerous. Still, the disease occurs in any age of infancy from a few months up. It is not so easily diagnosticated, because it is not expected and symptoms may not be pronounced. The brick-dust sputum may not be detected, for the infant does not expectorate. There will be dulness on percussion, and a crepitant râle, but the cough and sputum may not be characteristic.

I have recently treated a case of pneumonia, in the person of a child five years of age, in which the brick-dust sputum was never present, yet there was no doubt as to the diagnosis. In very old people pneumonia is liable to be latent or remain quiet for a considerable period of time, then develop without characteristic symptoms. I remember during one winter at the Louisville City Hospital we had several cases of chronic pulmonary phthisis in which pneumonia developed, which was only suspected, however, about the time that death took place, the pneumonic condition being confirmed by post-mortem examination. It was noticed that these phthisical patients

suddenly became very sick, with consolidation of the lung already present from tuberculosis. The diagnosis of pneumonia was not made clearly, we were not absolutely certain as to the immediate cause of death, until post-mortem revealed the fact that the patients died from pneumonia complicating the tuberculous disease.

Migratory pneumonia is one that travels from lung to lung, or from one lobe to another, more particularly the latter. It may begin in one certain spot in one lobe, in the base of the lung, for instance, spreading from there to the upper portion of that lobe, thence to other lobes of the same lung, or it may cross over and involve the opposite lung. When pneumonia involves more than one lobe, of course the danger is correspondingly increased, and the prognosis becomes more grave. The prognosis in all cases of pneumonia is grave. I do not mean by this that every case is liable to die, but it is no trifling disease, and not many mild cases are seen. When people have pneumonia they are generally sick; they go to bed and remain there, and are glad to do so. In young, vigorous adults, whose resisting powers are strong, the morbid elements are often successfully combated, but in the weak and debilitated the conditions are not so promising, and in any case it is a disease in which the prognosis must be guarded.

Pneumonia is one of the diseases in the treatment of which the profession cannot boast of any improvement as regards medication. Of course, there are exceptions, but taking all cases of pneumonia as they occur in practice, about one-fourth die, making the mortality twenty-five per cent. If cases are selected, considering only the young, vigorous adults, those in which complications do not occur, the prognosis of course would be much more favorable, with perhaps not more than a mortality of ten per cent., or even less; but taking cases as they occur in practice,—young and old, weak and strong, male and female, the debilitated, dissipated, the drunkard,—the prognosis will be about what I have stated, with a mortality of twenty-five per cent. The profession can boast of no improvement in the latter-day treatment of pneumonia; the outcome fifty years ago was just about as good as it is now, when they purged, bled, and vomited their patients, and did a great many other things that would seem to us to be rough and harsh.

The results of the treatment of this disease in private practice are better than in hospital practice, because hospitals get more of the

dissipated class, the worst forms, advanced cases, which in private practice would have been seen earlier in the progress of the disease. A fair estimate of the mortality of a given disease cannot be formed by taking only cases that occur in private practice, nor is it fair to take those occurring exclusively in hospital practice; both classes must be considered in order to arrive at an intelligent estimate. In country practice, where dissipation is not so common as in cities, with healthier subjects, better air, cleaner surroundings, etc., the prognosis is more favorable. I believe the prognosis in any case is better when the base rather than the apex of the lung is involved. The prognosis varies also according to the type of the disease which is prevailing. In this part of the country the prognosis is more favorable now than it was two or three years ago, and better now than it was during the prevalence of la grippe. So there are many things to modify the prognosis in pneumonia. The symptoms which should cause hesitation in making a favorable prognosis are a rapidly increasing pulse-rate, the strength of the pulse, and the presence of prune-juice sputum. I do not remember to have seen a patient recover in which that character of sputum was present together with the other complications mentioned. When there are no complications, of course there is always less danger. If a man has a good heart to start with, he has a much better chance of recovery. If he has a crippled heart to begin with, the chances are very much against him because of the fact that these patients generally die from heart failure, from asthenia rather than asphyxia, or from lack of proper aëration of the blood and over-distention of the right side of the heart.

Pneumonia may terminate in two or three different ways, but a termination by crisis on the seventh day is the rule. The crisis may occur on the fifth or sixth day, or it may be delayed until the ninth, tenth, twelfth, or even the fourteenth day. The disease, on the other hand, may terminate by lysis, or a gradual termination. When there is resolution by crisis, there is usually a dulness left in the affected lung, and it may be weeks before the lung sound will resume its normal resonance. After a man who has had pneumonia gets up and gains flesh and strength, there may still remain some dulness on percussion over the site of the disease. This dulness may last for a long time. This feature is often the cause of considerable anxiety, for fear of tubercular development. In some cases after termination of the active pneumonic process the lung may undergo fibroid degeneration

resulting in a contracted lung, or a cirrhotic lung, as it has been called by some authors. This is an unfortunate termination, but it does not necessarily mean that the patient will not recover, because he will sometimes become apparently well with the fibroid lung and will live for a long time afterwards. This fibroid degeneration is a favorable place for the development of other lung troubles, especially tuberculosis. This termination (fibroid) is, fortunately, comparatively rare. Just here I wish to insist that pneumonia does not of itself cause the development of phthisis, though in those cases where resolution is delayed for weeks it may act as a predisposing cause. Consumption develops only from the deposition of the tubercle-bacillus, from the development and growth of this organism in the lung, not from the pneumonic process.

The diagnosis of pneumonia, in ordinary cases, is comparatively easy. If there can be detected those sounds which are pathognomonic, crepitant râles and bronchial breathing, together with brick-dust sputum and a consolidated lung, anybody ought to be able to make the diagnosis. The clinical history is also usually very clear. A pneumonic process is more apt to be overlooked at the apex of the lung simply because its development is not suspected at that point. There is another reason also,—*i.e.*, that phthisis pulmonalis usually begins at the apex of the lung, and, of course, consolidation occurs at the apex as a result of phthisis just as it would from a pneumonic process; but the two diseases can be easily differentiated by remembering that in phthisis the development is usually gradual and entirely different from the prompt and sudden infection of pneumonia. By remembering these points the differential diagnosis between phthisis and pneumonia ought to be comparatively easy.

Cases of pneumonia that are most likely to be overlooked are those which develop in the course of chronic diseases, for instance, Bright's disease, diabetes, phthisis and other debilitating or depressing diseases, in which the patient is perhaps in bed, or is certainly not in a good state of health, and the physician is not looking for the development of pneumonia. In children the diagnosis is more difficult at first, because, as a rule, the meningeal symptoms are more pronounced, the pneumonia oftener at the apex, and one may be misled thinking of the meningeal troubles before development of lung symptoms of sufficient importance to indicate the diagnosis. In cases of pleurisy with effusion either complicating pneumonia or not, there

is of course some difficulty unless the physician is thoroughly conversant with the signs of these two diseases, there being no difference in the sound of the percussion note of pleurisy with effusion and the consolidated lung from pneumonia; but there is a difference in the sounds obtained by auscultation. In auscultating over the lung in pneumonia, bronchial breathing, the crepitant râle, increased vocal fremitus, bronchophony, etc., are detected; over that part of the cavity filled with the exudation no sound can be heard. The history of these two diseases is quite alike, both beginning with pain, cough, fever, difficult breathing, dyspnœa, etc., but with effusion there is an enlarged side, the mensuration is different, the intercostal spaces are obliterated, the percussion note absolutely dull or flat.

There ought to be no difficulty in differentiating between typhoid fever and pneumonia, but sometimes there is, because typhoid fever frequently begins promptly, sometimes with meningeal symptoms, with cough, and other evidences of lung disturbance. A line of symptoms may develop which will make the diagnosis a little difficult at first, but as the disease progresses, as either one of them develops, the line that separates them being quite distinct, and the course of the two diseases being far different, no trouble will be experienced in the differentiation.

Neurology.

NEURASTHENIA.

READ BEFORE THE LEBANON COUNTY, PENNSYLVANIA, MEDICAL SOCIETY,
MARCH 8, 1898.

BY AUGUSTUS A. ESHNER, M.D.,

Professor of Clinical Medicine in the Philadelphia Polyclinic; Physician to the
Philadelphia Hospital, etc.

NEURASTHENIA is sometimes called the American disease, and there can be no doubt that it is an exceedingly common disorder among us. While, perhaps, not exclusively a product of modern civilization, its prevalence has certainly increased with the keen competition and the striving for place and position and wealth that characterize our latter-day life. Figuratively, if not physiologically, the affection may be considered to be a derangement of functional nervous balance, in which the element of fatigue plays the most important part. The disturbance may be of any degree, from the slightest to the most extreme. Healthy function presupposes proper nutrition and a correct adjustment between cellular income and output, between nutritive pabulum and its assimilation on the one hand, and expenditure of energy and the production of waste upon the other hand. Disruption of this normal relation gives rise to manifestations of deranged function, and, if sufficiently long continued, to actual structural changes. It has been shown that there are distinct differences in the appearances of cells that have been engaged in active exercise and like cells when at rest. In addition to the alteration in function arising in consequence of changes in active cells, there are set free metabolic products that, if not removed, give rise to manifestations of intoxication. This is what takes place in neurasthenia, as well as in other of the so-called functional diseases, in a general way. For

myself, I prefer the designation nutritional. As I take it, the disturbance is essentially of this character, and the mode of treatment that is most successful is based upon this conception of the pathology. The deficiency may depend upon either the retention of excrementitious products, normal in quality but excessive in quantity, or of such products of totally abnormal quality, in either of which events it may be designated toxic; or an inadequacy of pabulum in either quantity or quality, when the condition may be viewed as one of inanition. From the foregoing considerations, it will appear that the popular terms nervous exhaustion and nervous prostration are not inappropriate.

As practically, with all of the disorders of the nervous system, the most potent etiologic factor is heredity, the neurasthenic parent being very prone to beget a neurasthenic child; and the influence of heredity is under such conditions as these accentuated by that of intimate association and education. Debilitating disease on the part of the parents is also not without an influence in predisposing to the development of neurasthenia in the offspring. The so-called neuropathic diathesis is probably of less importance in this connection, predisposing rather to the development of disorders of its kind, such as migraine, epilepsy, hysteria, and other neuroses and psychoses. An improperly trained, badly educated, and poorly disciplined child is more likely to develop into a neurasthenic than one in whom the opposite conditions have prevailed. Certain pursuits and occupations predispose to the development of neurasthenia, especially those demanding long hours, without sufficient intermission, and close application, with improper ventilation and supply of light. Positions of responsibility, of excitement, of tension, of importance, are favoring influences. It is difficult to decide whether neurasthenia is more common among unmarried than among married persons. Each class is exposed to peculiar responsibilities, but sexual excesses and improper sexual practices are not unimportant causes in both. On the other hand, sexual incontinence may be a causative factor. The disease is pretty evenly divided between the sexes; but it is peculiarly a disorder of the active period of life, although not unknown at the extremes of life. The most important exciting cause, however, is over-work, particularly in conjunction with under-rest. If emotional influences enter into the work, this factor is all the more important. Debilitating or exhausting disease may likewise act as a causative

agency. Traumatism, particularly if violent and abrupt and if attended with emotional concomitants, is a prolific cause. Accessory etiologic influences are the inordinate use of tea, coffee, alcohol, and tobacco. It can be conceived that the ill-advised or long-continued use of such stimulating drugs as strychnine or *nux vomica*, or *cinchona* and the like, may give rise to nervous exhaustion.

The disease may, and it ordinarily does, set in gradually and insidiously, but it may, as in the case of sudden traumatism or of profound emotional disturbance, set in abruptly. In either event the most distinctive symptom is an inability to perform the accustomed task, or an undue readiness of fatigue upon attempts to do so. Effort is soon followed by exhaustion. There is, beside, a general disinclination to activity, a "loss of ambition," as the patients are prone to state, a lack of spontaneity, a sense of languor, of more or less constant fatigue, undue drowsiness, with inability to sleep, such sleep as is secured being disturbed and harassed by dreams often of a disagreeable character, the patient commonly complaining of being more tired in the morning on arising than at night on retiring to bed. There is often a disagreeable sense of weight or pressure at the summit or the sides of the head or in the occiput and sometimes in the frontal region. There is also pain in the back, and there may be tenderness on palpation over the spinal column. "Nervousness" is often the patient's most prominent complaint, and on inquiry this is found to consist in tremulousness, sometimes spontaneous, sometimes induced, and when present spontaneously, accentuated by voluntary effort; it is most commonly present in the hands, but sometimes it is more widely distributed. Not rarely a sense of subjective trembling is complained of, although examination fails to disclose objective evidence of it. It may be that in some cases there is some such visceral commotion, but more likely the manifestation is a paræsthetic one, comparable with others that are observed. Patients sometimes complain of numbness, tingling, creeping sensations, burning and other paræsthesiæ unattended with objective signs. The patient is irritable, easily agitated, readily startled and frightened. He is emotional, even depressed, being moved to tears on slight or even no provocation. He is influenced by morbid fears, as of being alone, of being in crowds, of exposure in open places, that misfortune is impending, that he will not recover, and so on. This condition of melancholy may become a pro-

nounced feature, and in a predisposed patient it may pass over into actual melancholia, with delusions. The patient is likely to be introspective, to study himself with minute care, to analyze his symptoms with great detail, and he may become markedly hypochondriacal. There is general weakness, physical and psychic, but no actual paralysis or well-defined paresis. Like the nervous system, the muscles are readily exhausted and recuperate but slowly. The reflexes are in general irritable and marked upon first stimulation, but they are readily worn out and soon fail to respond. There is no objective derangement of sensibility. In addition to the paræsthesia spoken of there may be also hyperæsthesia. Eyesight and hearing are generally normal, but possibly unduly sensitive, and readily exhausted. Intellectual effort is difficult and not well maintained. There is no actual impairment of memory, but from failure to form proper conceptions there may appear to be such impairment. Function in general is wont to suffer. Appetite is impaired; the patients can take but little food, and this is but poorly assimilated. Digestion is enfeebled; constipation is commonly present, although sometimes there is diarrœa, and not rarely symptoms of so-called membranous or mucous enteritis. Nutrition necessarily suffers; flesh and strength are lost; color fades from the cheeks; palpitation of the heart appears; breathing is difficult; micturition may be increased in frequency; and the patient drags himself about wearily, with a most disconsolate air, presenting an aspect of utter hopelessness.

In diagnosis, care must be taken to distinguish neurasthenia from associated or secondary or even causative conditions. Organic and visceral disease of all kinds must be, and usually is, readily excluded, as well as all possible sources of intoxication, dietetic, occupational, medicinal, etc. Neurasthenia and hysteria are not rarely intimately associated, and care must be exercised not to fail to recognize the one behind the cloak of the other. While the hysterical may exhibit many of the symptoms of the neurasthenic, the latter will not present the sensory, motor, and psychic manifestations, paroxysmal and interparoxysmal, of the former. From true hypochondria and from melancholia neurasthenia is to be distinguished by the absence of established delusions. The neurasthenic can yet be made to realize that he does not actually suffer from the various diseases that suggest themselves to his irritable and susceptible mind, to his heightened imagination.

The prognosis in cases of neurasthenia is in general favorable, although the disease sometimes pursues a most protracted and obstinate course, and is occasionally ineradicable. Much depends upon the heredity, the surroundings, and the social condition of the patient, and his ability to carry out the details of treatment. As has been already implied, however, cases do sometimes progress to actual hypochondria, melancholia, and hysteria.

The principles that govern the treatment of neurasthenia are based essentially upon the conception of the pathology as already crudely sketched. Viewing the disorder as intrinsically one of disordered nutrition, with resulting cell-weakness and consequent irritability, the therapeutic problem consists in the restoration of the normal nutritive state; and this is best effected by removal of causative factors yet operative, the provision of rest for an overworked cellular organism, and the supply of a generous amount of proper nutritive pabulum. All sources of irritation, all disordered conditions of structure or of function, are to be removed or corrected; all excesses of body or of mind are to be avoided. The details of treatment will vary somewhat with the severity of the case. In the worst cases it is best to remove the patient from home, preferably to a well-equipped and suitably organized hospital, where he can be placed at absolute rest, and be given forced feeding, with the customary adjuncts, such as massage and electricity. The patient should be permitted to receive no visitors and no mail, and he should have communication only with the physician and the nurse, who should preferably be a masseuse. A vast deal more can be accomplished by thus isolating the patient than by permitting association with members of the family or with other patients in the wards of a hospital; though one is not always given the choice, and he must cut his coat according to his cloth. The basis of the diet should be milk, and for a time it will consist exclusively of milk. Some patients will contend that they cannot take milk, but a little persuasion and a little experience will demonstrate the contrary. It is best to give at first rather small quantities at short intervals. Thus, four ounces may be given every two hours, and when it is feared that milk will not agree with the patient, he should be instructed to sip it with a spoon. This will consume time, and will avoid the formation of large curds in the stomach. The milk may be boiled and taken hot or cold, in accordance with the taste and wish of the patient. If deemed necessary by the condi-

tions present, lime-water or sodium bicarbonate, or perhaps a little salt or nutmeg or Vichy, may be added to the milk, or this may be peptonized or sterilized, or perhaps modified in a variety of ways. The one point is that it shall be made assimilable and palatable. Even though the appetite have been poor, the patient will soon feel and show indications of hunger, and then the quantity of milk can be increased to six and later to eight ounces at a time. The feeding may be continued thus for several days or a week or more, when the diet is enlarged by the addition of a soft-boiled egg, together with perhaps a little stale or toasted bread and butter. Gradually, as appetite and digestion improve, tender meat, oysters, and fish may be added, and then stewed fruits, green vegetables, broths, soups, etc. To overcome the deleterious results that would otherwise arise from want of activity massage is employed, and to this the patient is gradually habituated. The manipulations are at first gentle and superficial, and continued for but a short time, say fifteen or twenty minutes; but gradually they are made more vigorous and deeper, and they are continued for an hour or an hour and a half. Made at bed-time, they are often conducive to sleep. Later, passive movements of the members are practised, and also Swedish movements. The patient is sponged daily with tepid water; but, as improvement begins to show itself, the temperature of the water may be gradually lowered. That is the most suitable temperature from which the patient best reacts, and it is determined by observation in each individual case. Electricity is a useful adjunct for exercising the unused muscles, although it is not improbable that its useful influence is not independent of the psychic effect. Usually, under the treatment thus outlined, the patient gains notably in weight, after perhaps a primary insignificant loss. With gain in weight comes also general improvement, and after a period of time, varying from six to twelve weeks, the patient can get up and about, and he will soon be able to resume his accustomed activity. The transition should, and is likely to, be a gradual one, and with the exercise of proper precautions the patients may be permanently cured. It must be borne in mind, however, that one who has suffered from an attack of profound neurasthenia is prone to break down if the pressure of work and the attendant conditions are favorable to such an occurrence, unless special precautions are taken. These consist essentially in the avoidance of excesses of all kinds, of emotional disturbances, worry, anxiety, grief, loss of sleep, irregular-

ity of habits, and indulgence in out-of-door exercise, the securing of a full amount of sleep, of sufficient and appropriate recreation, and diversity of occupation.

When the more rigid rest-treatment as thus outlined cannot be practically carried out, a partial rest-treatment may be employed, and this will suffice in the majority of cases. All etiological factors, and especially excesses of all kinds, are to be removed. Tobacco, tea, coffee, and alcohol in every form are to be abstained from, and all other extraneous sources of irritation and functional derangement are to be avoided. The patient should take a glass of milk every three hours, and, as his ability increases, the diet is to be enlarged by the addition of such articles of food as soft-boiled eggs, meat, fish, green vegetables, fruits, stale bread or toast, soups, broths, beef-tea, in addition to the milk. He is to retire early and to sleep late, say from six or seven in the evening until nine or ten in the morning. He may take his breakfast either in bed or at the table, in accordance with the circumstances of the individual case. He should rest after breakfast. He may subsequently go out of doors, but he should avoid fatigue. He lies down for an hour or two hours after the midday meal, and he may again go out afterward, again avoiding fatigue. After the evening meal he rests again or he retires to bed. Massage and electricity may be employed if possible, but a daily sponge-bath, with or without aid, should be insisted on. The bicycle or other form of systematic exercise may prove a useful adjunct. Out-of-door occupation is to be advised those who must continue at work. Obstinate cases of sexual neurasthenia in males may be greatly improved by shipping before the mast.

Of medicines, it may be said that no one possesses specific utility. The prescription must depend upon the symptomatic indications. Gastro-intestinal derangement may require the use of *nux vomica*, mineral acids, or pepsin; constipation, of the use of salines, aloes or cascara; sleeplessness and irritability, of bromides or sulphonal or trional; melancholia, of opium or morphine or codeine; anaemia, of iron or arsenic, and so on. Asafetida and sumbul sometimes prove of utility.

TRAUMATIC NEUROSIS AND A QUESTION OF DAMAGES.¹

CLINICAL LECTURE DELIVERED IN THE INSTITUTE FOR LEGAL MEDICINE AT THE UNIVERSITY OF BERLIN.

BY PROFESSOR FRITZ STRASSMAN,

Director of the Morgue and Professor of Legal Medicine at the University of Berlin,
Germany.

GENTLEMEN,—I have to-day a very interesting case to show you, in which my services in an official capacity were asked some time ago. The opinions then delivered will serve to show you some of the principles on which your judgment of such cases, in my opinion, will have to be founded. They are, as a rule, extremely difficult cases to deal with, and in the hurry and bustle of modern life the nervous excitability that this develops and the autosuggestion that is the consequence of the supreme self-consciousness and introspection of the time is making them ever more and more frequent.

Seemingly slight accidents are followed by nervous conditions, partly the consequence of that indefinite cause concussion of the central nervous system, mostly due to disturbing influence that any shock, however slight, may have upon the deteriorated nervous systems of individuals who are the atavistic representatives of degenerate ancestors. In our day this chapter of the traumatic neuroses has assumed an importance that must make it interesting to all, but especially to such as, owing to their duties as legal experts or their connection with damage suits for accidents or accident insurance companies, will be called upon to form and give definite opinions in the matter.

Our patient is a man of thirty-five years, a travelling salesman, who twelve years ago, in June, 1887, while riding as a passenger on a train of the Prussian Railway, was in a collision that occurred at Wannsee, a suburb of Berlin. According to his history, he was un-

¹ Reported by James J. Walsh, Ph.D., M.D.

conscious for some time, and suffered some superficial wounds. His arms were bruised, his hands and face were scratched, and black and blue marks made their appearance afterwards on his trunk, especially on his right side. He was brought first to the Augusta Hospital, where his scratches and bruises healed without complications. Certain subjective symptoms remained, however, even after all the objective symptoms had disappeared. He was nervous and easily fatigued. He complained of not being able to make much exertion, and of utter inability to do any serious work, though, according to his history, he had been a faithful and conscientious workman before the accident.

The clinical conclusion, as recorded at the Augusta Hospital, was as follows: "The patient suffered during the collision from concussion of the central nervous system, of which the nervous symptoms, the neurasthenic condition, and the asthenic state he is now in are a consequence." After having been at the Augusta Hospital for some weeks, but without notable improvement of his nervous symptoms, he was under treatment at several sanatoria for nervous diseases, but without benefit. He then applied for the pension that, according to German law, is paid by the railroads to persons injured in railroad accidents. The amount of this is, as you know, determined by law in accordance with the extent of the injury received and the wage-earning capacity that has been damaged.

At the formal examination to decide this the patient complained of a dull sense of pressure in the region of his forehead, as if the scalp were tightly stretched over the underlying skull; at times there were flashes before his eyes, especially what he describes as falling or raining pearls, and a vibratory feeling as if a steam-boat were at work. When he attempted to read, the letters swam before his eyes; he was often hoarse, his memory was weak, and there were dull pains in his back, which radiated into his arms and legs, so that he often had the sensation of his extremities being asleep. Often, when he attempted to walk, he had the feeling that he was fastened to the ground, unable to lift his feet; often, when lying down, he experienced the sensation of being stretched out,—i.e., as if some invisible force were, by means of impalpable cords, pulling him out, making him longer. On awaking from sleep he often felt as if he could not move his legs.

At times he had the tremorous feeling that he saw people when

they were not present, or he saw smoke when there was none. He was given to sleeplessness, and was easily disturbed by the slightest noise. Sudden noises, or the loud bustle of the street, made him nervous and excitable, and put him at times quite beside himself. He often had the feeling of the blood going to his head, and at times, when it did, it seemed to him as though he lost his senses for the moment.

The medico-legal expert who examined him at the time found a relaxed muscular system, a flushed and warm condition of the skin of the face, an insecure gait, a tendency to titubation when the eyes were closed, absence of the patellar reflexes, diminished sensation on the left side of the body, regular and normal pupillary reaction in both eyes, some congestion visible to the ophthalmoscope on the eye ground, and a congestive cyanosis, though not very marked, of the mucous membrane of the mouth and pharynx. The pulse was rather slow, and frequently missed beats. The diaphragm was half a centimetre lower on the right, and there was a cuirass-like lifting of the anterior thoracic wall with each movement of inspiration.

The conclusion expressed by the medico-legal expert after his examination was that the patient, in consequence of the accident, had been put into a condition of health in which for a time, the length of which could not be foreseen at the moment, he was incapable of making a living for himself. He considered further, when directly questioned, that, under the patient's circumstances, he had a right to all the expenses of medical treatment so far incurred, and to whatever extraordinary expenses should be hereafter deemed necessary for his proper care and treatment while his nervous condition continued. Besides the full salary he was getting at the time of the accident, he was adjudged to have a right to three marks a day (seventy-five cents), because of the condition he had been put into by the accident and the additional care this condition required.

The patient received his damages and the pension mentioned for eight years, until the fall of 1896. Then he got married, and rumors reached the officers of the railroad company that he had taken to drinking. As a result a new medical examination was asked for by the company, and for some weeks the patient was under observation in a private sanatorium here in Berlin.

According to the clinical record then made, the patient complained of constant feeling of pressure within the skull, at times dis-

tinct pain in the occipital region. From time to time he had the feeling as if something in his head gave way, as if something burst, or as if sand was rolling about within his skull. He still had almost constant weakness of his legs, which was increased after he experienced the peculiar feelings mentioned above in his head, such attacks being at times preceded by a feeling of anxiety, with a sense of compression around his heart and in the heart region. He often suffered from a rush of blood to the head, and had constantly the feeling that a cold object was being pressed against his forehead. He suffered from melancholic depression of spirits, with outspoken tendencies to cry without reason, and was extremely irritable and excitable. Words often failed to come to him when wanted, he often lost the thread of the conversation when talking even with friends, and could not keep up a continued conversation at all with even comparative strangers. He had at times attacks of doubting, the condition that the French call *folie du doute*, during which he scarcely trusted the evidence of his senses and doubted of facts immediately after their occurrence, though he had seen them happen. At times he saw figures and colors and heard voices. He constantly slept badly and had unpleasant dreams. His voice had become higher pitched. His left leg was weaker than his right. His feet were constantly cold, and his hands suffered from the cold in the winter time.

The objective symptoms brought out by the examination were: numerous dilated blood-vessels on his face, twitching of the upper eyelid when he brought his lids together suddenly and forcibly as in winking, contracted pupils that react normally to light and to accommodation, tremor of the tongue, slight vibratory tremor in the outstretched hands, slight swaying when he stood up with heels and toes together and closed his eyes. In his left arm and left leg the muscular power is diminished, the patellar reflexes are very much diminished; the pulse is regular, medium full, and varies between one hundred and one hundred and twenty to the minute. There is strongly marked dermography; that is, there exists an urticarial, irritable condition of the skin and of the vasomotor mechanism in it, so that the irritation of a pencil or pointed piece of wood used to trace letters on the skin is followed by wheals that persist for some time.

The vocal cords do not close completely, and there is anomalous action of them in phonation. Alimentary glycosuria is present. Physically everything else is normal, so I spare you the account.

As to his psychic condition, it was noted that he was in a persistent state of excitation, always on the verge of tears, and shedding them at times abundantly; his utter want of serious reflection often made itself manifest. For simulation or for a tendency to drink to excess the examination gave absolutely no grounds for suspicion.

The medical opinion given after this investigation of the case was that the patient, in consequence of the railroad accident, was suffering from hystero-hypochondria. In his present condition he certainly seemed to be able to take up some light employment, to do the work of an overseer or sort of day watchman in a large store, to help in a small business where transactions were simple and uninvolved, or to copy in limited amount. It was a question, of course, whether he would be able to find a suitable position of this kind, but it certainly seemed that his total wage-earning capacity was not gone. In all justice, perhaps, three-fourths of his wage-earning ability was no longer utilizable, so that seventy-five per cent. of his former salary should be paid him and nothing additional for medical expenses, etc.

Against this proposed reduction of his pension by more than twenty-five per cent. the patient took an appeal and presented to the court the opinion of the medical directors of the sanatorium for nervous diseases at Lichtenfeld, near Berlin, where he was under observation for several weeks, some time after the examination made at the instance of the railroad company. These medical men found that the patient complained of a host of symptoms mostly merely subjective, which varied from time to time in intensity but never left him, and that they considered that these symptoms made it impossible for him to find employment in which he could seriously think of supporting himself. They especially were impressed by his complaint of persistent headache, mostly in the frontal region and combined with cold sensations, feeling of weakness in the sacral region, in his left arm and leg, the sensation that these parts were asleep, the sense of anguish with compression in the heart region, the cold hands and feet, the occurrence of black spots and blue smoke, with the rain of pearls before the eyes and the hallucinations as to seeing figures of other kinds, all of which added to his condition of anxiety. Then he had hallucinations of hearing, and heard the voices of people whom he knew to be long since dead. He slept badly, and was always tired in the morning. When he attempted to read, after from

ten to fifteen minutes the letters swam before his eyes. He further complained of urgency of urination, his sexual potency was diminished, he was inclined to tears, he grew uneasy and frightened whenever he was in a crowd, and had other psychic manifestations of a like character.

The examination showed objectively a relaxed muscular system, piteous facial expression, as if of constant suffering, flushing of the face and some redness of the eyes, distinct and concentric narrowing of the field of vision in both eyes, marked tremor of the tongue, hesitating speech, often at a loss for words, the effort to find them being accompanied by sympathetic movements of the face and of the arms. The pulse frequency varied between eighty-four and one hundred and eight beats to the minute. The hands always feel cool and moist and are persistently tremulous. The patellar reflexes are decreased. Romberg's symptom—swaying with closed eyes—is present to a marked degree. Dermography is present, and there are a number of points painful to pressure to be found. Sensation was so far diminished that two pin-punctures made at the same moment were felt as but one, though the distance between the points of puncture was considerable.

The patient was constantly in a depressed state of mind, was quiet and occupied with himself, sat by preference in solitude without seeking to occupy himself, showed no interest in his surroundings, burst into tears and audible sighs often, and had from time to time the idea that he was being persecuted; he was slow and labored in thinking, made mistakes in the simplest reckoning, though he had had a good common-school education, and often forgot things that were of importance to him and were of recent occurrence.

These observers absolutely excluded all idea of simulation in the patient's case, and found his condition a genuine pathological one; especially was his psychical state such that they judged him to be totally incapable of making a living for himself, and stated this as their opinion to the court.

With medical expert opinions that were thus somewhat contradictory, the court referred the matter to its own medico-legal experts, and so the case came to us at the Institute for Legal Medicine. We shall have to decide which of the opinions the court should adopt.

My preliminary examination of him some days ago gave me the same impression as had been produced on the others. His complaints

are practically the same as those he made at the other examinations. He still complains of continuous dull headache and peculiar feelings in his head, as if something like a bubble burst within it every now and then. He occupied himself lately somewhat with gardening, but could stand scarcely any fatigue. When he attempted to read, the letters danced before his eyes. His feeling of depression grew worse and worse. He slept little, and his sleep was disturbed by bad dreams, in which his life seemed to be in danger. It was hard to get to sleep, and he often broke out into tears in his sleep. When he walked it was not long before his left leg began to drag; his left side was generally paretic. He often had oppressive feelings around his heart and often suffered from attacks of vertigo, so that he dared not go on the street alone. His sexual potency was diminished.

During my examination I noted objectively his tendency to tears, and he wept and wrung his hands at times. He repeatedly gave vent to such expressions as: the railroad company was persecuting him to death; they followed him even into the bosom of his family; they were collecting documents against him. His replies were slow and labored, and his voice was high pitched,—a falsetto.

Our examination of him now will be mainly confined to objective symptoms. His face is flushed and shows dilated blood-vessels; there is tremor of the upper eyelids and of the tongue. When I have him stretch out his hands you notice a fine vibratory tremor here, too. When he stands with feet together there is moderate swaying. His knee-jerks are diminished and asymmetrical, that on the left being scarcely demonstrable. When I test his sensibility he says that he feels the stick of a pin much less on the left than on the right. That objective anaesthesia really exists here, his almost impassive condition and the absence of reflex when I suddenly stick him in this region while talking to him seems to show. While he confesses to feel the slightest touch of a camel's-hair brush on the right, he misses all gentle touches on the left. As I draw the end of this penholder over his skin you can see the hyperæmic mark that develops in its track, which after a few minutes becomes an urticarial ridge. Here on his back I can make letters by means of this factitious urticaria that remain visible and palpable for some time. This is a very good example of the disturbance of cutaneous vasmotor mechanism which has been called dermography,—skin-writing. Examination of his eyes shows that the pupils are normal and react promptly to light and

accommodation. There are no abnormalities in the eye-ground. His field of vision is concentrically narrowed to a considerable degree. Its exact limits you can see on this chart that I pass around, but you can judge of its great limitation by how soon my watch, when moved in his field of vision becomes according to his declaration invisible.

His pulse is regular and one hundred and twenty-two to the minute. Its rapidity is probably partly due to the excitement of examination, but this very tendency to increased frequency, that persists when sufficient time has elapsed for the excitement to have subsided, is characteristic of neurosis. The pulse is too frequent now to hope to develop Mannkopf's symptom,—namely, the increase of pulse frequency when pressure is made over an avowedly tender spot. During my previous examination I think that at an especially calm moment I succeeded in bringing it out.

We have, then, subjectively, sleeplessness, the headache with sense of pressure, the tired feeling, the vertigo, excitability and loss of appetite and sexual potency, with neurasthenic hypochondria, that are the distinctive symptoms of traumatic neurosis. We have objectively the concentric narrowing of the field of vision, which most specialists agree cannot be simulated, the heart palpitation and increase in frequency of the pulse, the tremor and tendency to incoordination, the tendency to blush, and the dermography that indicate disturbance of the vasomotor nervous mechanism,—we have all of these to assure us that it is not simulation we have to do with here, but a genuine neurosis.

It has been the custom to think that many of these cases were simulation, but as our knowledge of the functional neuroses has become more exact in the last ten years, our suspicions as to simulation have been more and more dispelled. In some very interesting cases where simulation was judged to be present some years ago, the further course of the disease has shown undoubtedly that the condition was pathological. I prefer to think myself with Hitzig that it is impossible for the class of people to which such patients often belong to simulate for a length of time, with any probability of deceiving a careful physician, that after all perfectly distinct symptom complex, neurasthenic hypochondria.

We have in this case, owing to the legal complications, a very full and authentic history of the case. The consentaneity between the earliest stages in the time immediately succeeding the accident down

to the present, ten years afterwards, is very evident. The examination made in the interval two years ago is but confirmatory of this. Whatever differences there are, are only such as could be easily explained by the fact that the manifestations of traumatic neurosis vary within certain limits, and so make certain symptoms much more prominent for the patient than they were before. This persistence of the clinical picture of the case is the best evidence that we have not to do with simulation.

Judging the case on its merits, then, we can only say that he deserves the full amount of the pension allotted to persons unable to work, for while he is able to do a certain amount of work, perhaps, that is of such a limited character, and would have to be permitted under such indulgent conditions as to time, that it is extremely doubtful if he could secure an employer on his terms. Meantime, he has found a wife to take him, and it is possible that he may find an employer, but until he does his pension should be the full amount for a man unable to earn his living.

It is the fact of his getting married and the report that he was drinking that led to the railroad company's reopening the case. His wife, with whom I've had a talk, is an old friend whom he has known from childhood. The death of relatives had left them alone in the world, and so they got married; she seems to have the best of intentions to take care of him. She called my attention to the fact, which could easily be substantiated, that her husband, because of his extreme sensitiveness to the noise of the street, had had double doors put into their dwelling. She also told me that it was almost impossible to walk with him on the street, owing to the state of excitement that developed whenever anybody touched him.

As to the report of drunkenness, it seems to have been absolutely without foundation. There seems to be no trustworthy evidence that he ever indulged in spirituous liquors to excess. This is important, as a condition similar to that in which he now is at times develops as the result of alcoholic excesses over a long period. It has been noted that after an accident a condition like this developed which seemed to be a traumatic neurosis, but the symptoms of which could really be traced to chronic alcoholism before the accident. In such a case, then, the question of damages is modified by the amount of the neurotic symptoms for which the patient's former habit is to blame. Here no such allowance will have to be made.

I have more than once had employees to examine for advancing neurasthenic symptoms that finally prevented them entirely from working in whom the want of a complete history left me entirely in the dark as to the origin of the affection. Where the labor required was exacting, the presumption was uppermost that it was due to the employment. Later the personal history, with the knowledge of the abuse of alcohol, made the case clear.

Here we have no such history. While the patient's psychical condition at present would serve to show very probably that there always existed a certain lack of absolutely stable psychic equilibrium, that he was more liable than most people to the development of neurotic symptoms, still there is no history of any symptoms beforehand; he was simply a faithful workman. There is every reason to think that but for the shock to his nervous system at the time of the accident he probably would have been able to continue for years in the regular fulfilment of his duties. There is, then, good reason for the payment of the pension that we have spoken of.

I may add here that the sooner the matter is definitely settled the better for the patient. The longer, especially at the beginning of such a neurotic state, that a patient is kept on the tenterhooks of expectation, with his attention directed to his every symptom and the consciousness that he is the subject of distrust and observation, the more permanent are likely to be the effects upon his nervous system. It is probable that if the matter could be settled at once, many cases that now drag out for years would gradually grow better, and the suggestive influence of a favorable decision would co-operate not a little to produce such an eminently to be desired result.

As it is, often owing to prejudice in the matter, rapid improvement of the patient after a favorable decision is looked upon as a sign that simulation has been at play. There is no doubt that the intense self-conscious attention of the patients to their symptoms leads to a more or less unconscious exaggeration of them, but this is in itself a sign of the hypochondriac condition that results from the neurosis. Do not be too ready to believe in simulation in a case because you find some exaggeration of symptoms, and in general remember that the serious mistakes in such cases have not been made by giving credence too easily to successful simulation, which is extremely rare, but to the judging a poor neurotic patient to be an interested simulator. A serious mistake, indeed.

OPTIC NEURITIS AS AN EARLY SYMPTOM IN NERVOUS DISEASES.

CLINICAL LECTURE DELIVERED AT THE PHILADELPHIA HOSPITAL.

BY JAMES HENDRIE LLOYD, A.M., M.D.,
Neurologist to the Philadelphia Hospital.

GENTLEMEN,—I wish to show you to-day two interesting cases in young persons which commenced with involvement of the optic nerves. These cases presented themselves at the clinic with this one initial symptom prominent above all others,—i.e., rapid involvement by inflammatory action of the optic nerves, leading to total blindness. The first case is that of a boy sixteen years of age, while the other is in a young man of about twenty-five. These patients present themselves to our study with the one symptom in common, and we have not much else as yet upon which to base the diagnosis. We must, therefore, begin with this one symptom, and by careful investigation and cross-examination we must search for other symptoms and find out the cause and the exact pathological condition. This certainly is a very interesting pathological problem, and one which we owe to the richness of this clinic, for probably there is scarcely a hospital in this country that would enable a lecturer to pick out at one and the same time two such extremely rare cases in young people. I saw one of these patients for the first time two weeks ago and the other one only yesterday afternoon. I shall first take up the case of the boy and see what I can make out of it. I do not now know what is the matter, but hope to find out to-day. Before going further I will give expression to a truism, and say that the optic nerve does not degenerate in an inflammatory process unless there is some radical cause for it. These degenerations of the optic nerve are of extreme importance, and there is always some hidden cause for the condition. It is this that it is for us to find out. The conditions of the optic nerve are important neurological studies, and although they are properly

given over to the ophthalmologists in hospitals like this, where we have to divide up our work, it is essential for the neurologist to understand them thoroughly.

Let me remind you briefly of the anatomy and pathology of the optic nerves. The eye, retina, and optic nerve are not peripheral organs in the ordinary sense, for this nerve differs entirely from all the other nerves of the body, unless probably the olfactory, as is shown by its development. The eye is developed from the same epiblastic tissue as the brain cortex. There are two layers of nerve-cells, or neurons,—the superficial ganglionic and the deep ganglionic. In all the peripheral nerves there are two sets of neurons, the peripheral and the deep. This is also so in the retina, the superficial being in contact with the deep set. The cell bodies represent the trophic centres of the optic fibres. They are the nutritive centres of the neurons. The optic nerve-fibres do not grow from the brain towards the eye, but the vast majority of them grow from the eye towards the brain. Descending degeneration is not a process that occurs from the brain towards the eye, but occurs towards the brain, according to the Wallerian law. The degeneration may in some cases be present early in the optic nerve, as viewed by the ophthalmoscope, before the central nervous system is seriously involved. In certain diseases of the nervous system the optic nerve degeneration is a common forerunner or accompaniment. In locomotor ataxia especially we may have degeneration of the optic nerve as an early symptom. This has been a source of speculation to the pathologist. In degeneration of the posterior columns of the spinal cord we often have a similar condition in the optic nerve, and syphilis is the most common cause of this.

Now as to the causes of optic neuritis. Probably the most common is brain tumor. Possibly eighty per cent. of all brain tumors show at some time or other inflammation of the optic nerves. If you have a suspicion of brain tumor you must assure yourself of the condition of the optic nerves. On the other hand, if you find inflammation of the optic nerves as an early symptom, you must assure yourself as to possible symptoms also of brain tumor. The old theory was that the brain tumor filled up the intracranial space and caused obstruction to the circulation and the lymphatics, and this in turn produced congestion and inflammation or atrophy of the optic nerves. This would produce choked disk or optic neuritis. This is a purely mechanical

theory, and is not a satisfactory one. A more recent theory is that the brain tumor causes a damming up of the lymph- and blood-vessels, and thus interferes with nutrition. The result is that the products of retrograde metamorphosis, being unabsorbed, act as irritants, and cause inflammation and degeneration of the nerve-tissue. As I have already said, optic neuritis is a very common symptom of brain tumor, and the most common brain tumors that cause this condition are those that occur beneath the tentorium. The primary distribution of the optic-nerve fibres is largely in the mid-brain, and this is probably the reason why cerebellar tumors cause optic neuritis so promptly. The tumor, making direct pressure upon these fibres, is more prone than tumors in any other region to cause irritation of the nerve. Brain tumors in the anterior frontal region cause optic neuritis more rarely, although it would be an error to say that they never cause it.

Next to brain tumors the most common cause of this condition is syphilis. In some cases specific inflammation occurs in the membranes at the base of the brain, and the optic nerve is involved. The optic nerve is in close proximity to other basal nerves, such as the third, fourth, and sixth, and all these nerves may give distinct symptoms. Consequently, in a basal syphilitic meningitis we might have optic neuritis, but we would be likely to have symptoms of involvement of other cranial nerves along with it. Syphilis may cause degeneration in another way. In locomotor ataxia, a disease which is not due to a distinct syphilitic meningitis, the optic nerve-tract is very likely to degenerate, and this is called primary optic atrophy. It is really a parenchymatous degeneration, and occurs from the same cause that acts on the fibres in the cord. In such cases the common cause is a blood-poison. The inflammation does not spread from the cord to the optic nerves, because there is no direct anatomical connection between the two.

The third most common cause of optic neuritis is lead-poisoning, of which we see many cases in this hospital. Sometimes this condition is not altogether recognized. One of the cases before us presents this problem. Chronic lead poisoning causes inflammation of the optic nerve, and this inflammation usually begins behind the eye proper and is called retrobulbar inflammation of the optic nerve. Such an inflammation may be caused by other forms of acute poisoning, such as tobacco, alcohol, quinine, the bisulphide of carbon,

etc. These poisons all act as slow nerve-destroyors, and may ultimately cause complete destruction of the optic nerve. Under the microscope there is found a proliferation of the blood-vessels and connective tissue as well as a destruction of the medullary matter and axis cylinders.

The fourth most common cause of this condition is Bright's disease. Chronic interstitial nephritis will primarily cause an inflammatory condition of the retina and of the optic nerve. The optic neuritis due to Bright's disease is generally characteristic, and is usually recognizable with the ophthalmoscope. It is associated with evidence of great degeneration all through the eye ground and retina and with hemorrhages. You do not usually see this in the optic neuritis due to lead, syphilis, or brain tumor. It occasionally happens that you do not see all of these distinct retinal changes, but you may find simply an inflammation of the optic nerve. Now, the question arises, Is the condition in this patient whom I show you due to a brain tumor or some form of syphilitic infection? If syphilitic infection, is it associated with posterior sclerosis or locomotor ataxia? Is it due to lead poisoning? Is it due to Bright's disease? We will first determine whether this young man has a cerebellar tumor, of which I have seen several cases here within a year. I had a case here last year of cerebellar tumor with optic atrophy and blindness. In cerebellar tumor one of the commonest symptoms is headache; other symptoms are a peculiar staggering gait, vomiting, attacks of vertigo, loss of consciousness, and convulsions. This boy says he has never had a headache, and, excepting the blindness, there is really nothing the matter with him. His knee-jerks, however, are slightly diminished. They are increased by reinforcement. We have, therefore, no symptoms of brain tumor, no ataxia, no swaying while walking or standing, no paralysis, no tremor, no headache, no vomiting, and the patient has never had convulsions. When I first saw this case I suggested the possibility of some beginning cerebellar lesion, but I do not believe now that it is a cerebellar lesion, as I can hardly conceive of a patient going blind because of a cerebellar lesion without some other symptoms. Now, as to syphilis, in some forms of syphilitic infection we have the sensory type of locomotor ataxia. In this type we may not see any of the symptoms of posterior sclerosis except the sensory symptoms. In locomotor ataxia ordinarily there are very marked motor symptoms, such as

swaying movements and an ataxic gait. We may have, on the other hand, a type of locomotor ataxia in which we do not observe decided motor symptoms. This type is characterized by very early primary optic atrophy and violent fulgurant pains. The optic degeneration comes on before the other characteristic symptoms of locomotor ataxia. Buzzard, of England, has put on record a case in which the optic atrophy began fifteen years before the appearance of the other symptoms of tabes, and Gower has reported a case which began twenty years before the ataxia. I have in my series of cases seen similar ones. It is extremely improbable that this boy will develop the ordinary disease known as locomotor ataxia, as he is so very young. I never heard of a case of locomotor ataxia caused by the poison of syphilis occurring in a boy of sixteen. Of course, such a thing is not impossible. This patient may have had syphilis himself, or he may have had hereditary syphilis, but we are unable in any way to determine in his case the presence of syphilis. He has not Hutchinson's teeth, and there is nothing characteristic about the mouth. He states he has never been exposed to syphilis. He has not a primary optic atrophy, such as occurs in locomotor ataxia, but he has a form of degeneration of the optic nerve which follows acute inflammation. He has one rather suggestive symptom, and that is a slight weakness of the bladder, which is sometimes a very early sign of locomotor ataxia and is very suggestive of posterior sclerosis. In addition, he has noticed sexual impotence. With the symptoms we have thus far mentioned we have but one thing left to search for, and that is the pains peculiar to locomotor ataxia. I am unable to obtain any history of these pains in this case. As a rule, these pains, if present, are very severe and are unmistakable. The urine is negative. In spite of the negative history, I am inclined to believe the case is one of latent syphilis, in which the poison has attacked the basal membranes and the optic nerves.

The second case is in a young man, twenty-five years of age, who complains of failure of vision, and weakness in the lower extremities. His family history is negative, and he himself has always been healthy until his present trouble began, which was only a few months ago. At that time he noticed that his sight was becoming a little hazy. At times he could not see well, and a little before this period he had noticed some evidence of sexual impairment and some paresis of his bladder. There is a distinct history in the case of exposure to

lead, as the man has been a wagon-painter for four and a half years. You are often told that in lead-poisoning the important symptoms are abdominal pains and wrist-drop, but you must not forget that lead is a more or less universal poison to the nervous system, and may cause a great variety of symptoms. It can poison the brain centres as well as any nerve-tract or fibre in the body. It can also poison the kidneys and the stomach. You may see striking brain symptoms, such as headache, convulsions, and delirium, denoting a lead encephalopathy. You may see paralysis of the optic nerves as well as of the radial nerves, and you may observe a distinct and peculiar tremor. In addition, you may see a form of gait closely simulating that of locomotor ataxia, due to involvement of the sensory nerve-endings. This is called pseudo-tabes. You may see this ataxic gait, as well as muscular atrophy in the legs and abolition of the knee-jerks. Then, too, you may see the evidence of interstitial nephritis, or chronic Bright's disease, as well as the blue line on the gums; and, finally, a certain amount of anæmia and dyscrasia that is quite characteristic of lead-poisoning. This young man has post-neuritic optic atrophy. The optic disk is contracted and white, and the outlines are irregular and hazy. He presents the type of optic atrophy that comes on after a neuritis. There is probably an inflammation back of the eyes, a retrobulbar inflammation. This is the prominent symptom, optic atrophy following optic neuritis. The kidneys in this case are not involved, but one must remember that every case of lead-poisoning does not present inflammation of the kidneys. This patient has none of the symptoms of cerebellar tumor. He has only had slight headache, but no vomiting, no peculiarity in gait, no vertigo, and no convulsions. He has perfect use of his extensors. I have seen cases of lead-poisoning which have shown atrophy of the shoulder girdle muscles. There is no paralysis in this case of the third, fourth, fifth, sixth, seventh, or twelfth nerve. The man is slightly ataxic and has a slightly enfeebled gait. When he stands with his feet together you notice that he has a tendency to claw the earth. This is seen in locomotor ataxia and in pseudo-tabes. He has no history of pains in his legs. His patellar reflexes are abolished; and, on the whole, I am inclined to think his case is one of beginning lead-poisoning. This is indicated by the fact that he has the distinct form of optic neuritis that we see in lead-poisoning, and he has, moreover, a distinct history of exposure to lead. These painters poison them-

selves in a very simple way. They do not inhale the lead; they eat it. It gets under their finger-nails and in the pores and creases of their skin, and in that way it is carried into the mouth when they eat, and thus into the stomach, practically from want of cleanliness. In a lead-factory it is different, for there the air is loaded with lead dust, and this is inhaled. But even then it is probably also mixed with the saliva and swallowed. Lead nearly always enters in this way,—*i.e.*, through the *gastro-intestinal tract*. This man, to be sure, has not the history of ordinary lead-poisoning, and only shows a slight purple line on the gums. But although the case is not altogether clear, the history is very significant, and I think I am justified in saying that it is a case of lead-poisoning involving primarily the optic nerves.

Surgery.

GENERAL REMARKS UPON RECTAL DISEASES, WITH ESPECIAL REFERENCE TO THE TREAT- MENT OF FISTULA.

BY JOSEPH M. MATHEWS, M.D., LL.D.,

Professor of Surgery and Clinical Lecturer on Diseases of the Rectum in the Hospital
College of Medicine, etc., Louisville, Kentucky.

RECTAL diseases, as is now well known, are very common, affecting all classes of people, from the infant to the aged, women as well as men, rich as well as poor,—a class of diseases that has been neglected, indeed, from time immemorial; every other part of the body received attention in a scientific way from the surgeon except this part. Up to about twenty years ago nearly every patient suffering from a disease of the rectum was referred, even by the reputable medical practitioner, to quacks. They had complete control of this branch of medicine and surgery; but, strange to say, not much is due them in the matter of investigation, because their practice was not based upon any scientific principle; very few of them knew any anatomy; they knew nothing about the principles of surgery; but they saw in it a chance to make money simply because the physician and surgeon either did not care to operate in this line or had never investigated it.

The rectum is not only the seat of many diseases, but diseases that threaten human life, diseases fatal in their character, some of the most tormenting, painful, distressing ailments that you can find in any other part of the body.

But things have changed. In every city of the United States now of any size will be found men giving special attention to diseases of the rectum, eminent surgeons who have made it a study. Then,

again, general surgeons, whether in the country or in the city, have paid as much attention in recent years to this class of work as they have to other portions of the body. Hence, there are many good practitioners in this line, and fortunate it is that these affections have been wrested from the domain of the charlatan, the quack, and placed where they belong,—viz., in the legitimate field of medicine and surgery.

Nothing is more common in a surgical way, for instance, than hemorrhoids. It is the rarest thing to find a person who will go through life without some affection of the rectum, either a man or woman, and especially women, from the fact that they are child-bearing, producing, by pressure of the child in *utero*, hemorrhoids. Often during pregnancy and other conditions the physician will be called upon to treat a woman for some rectal affection; it being paramount in her case to anything else, from the fact that it is very painful, distressing, and she fears will interfere with the birth of her child. In men we will meet with quite as large a percentage of rectal affections as in women, and to offset the child-bearing function in the latter we have the prostate as a causative factor in the former. Irritation of the prostate, gonorrhœal or otherwise, stricture or any inflammatory condition of the urethra, produce many affections of the rectum, especially hemorrhoids, proctitis, etc.; but in the vast majority of cases of diseases of the rectum, fistulæ, hemorrhoids, etc., the physician will be unable to find any cause. He may think that theoretically he has learned all about it; but when he meets young ladies and young gentlemen who are not given to vices or bad habits who have these affections, I say it is a difficult thing to tell what is the *fons et origo mali*.

If I were asked what was the main cause of hemorrhoids, for instance, I would be at a loss to answer, because I have just as often operated upon perfectly healthy people for this condition as I have upon those who were unhealthy.

Aside from a special diathesis, if I were asked what was the cause of fistula in *ano*, I would be at a loss to say. For either one of these two diseases I would have to answer, “I do not know.”

We give these people special directions as regards movement of the bowels, hygiene of the same, regular habits, etc., but those who observe such directions very often have rectal disease. Take, for instance, the negro race; how often does the surgeon operate upon negroes for hemorrhoids? Very seldom. They are singularly ex-

empt, and yet their habits are such as would be conducive to the hemorrhoidal condition. On the other hand, fistula predominates in the negro race; but it is more or less a question of heredity, the tuberculous diathesis or habit.

Race has much to do with all kinds of surgical affections, and especially is this true with diseases of the rectum. Van Buren says that in his early life he had much to do with the Indians, living largely with them, and that he never saw an Indian in his life, male or female, that had hemorrhoids, which should be a lesson to us, that life in the open air, the exercise that they take, the freedom from many things common to those in fashionable life, occur to us.

The physician in practice will not infrequently meet with rectal affections even in the infant. First, for instance, I would call attention to the fact that an infant may be born without an anus, without a rectum, or with the rectum diverted out of its natural channel. A malformation of the rectum may exist, and if the physician is not on the alert and quick to comprehend, the child will die because there is no outlet; but by a little surgical procedure on his part, he saves its life, because it is healthy in every other particular, by making for it a natural anal outlet. Again, children are especially liable to have prolapse of the rectum. This is due to the fact that the anatomy of the rectum, as far as its normal conformation is concerned, is not perfected,—the sacrum having but little groove, the mucous membrane being loose or movable, and straining that is natural to infant age causes the bowel to protrude, and there is a prolapse. It is not a simple thing; it will bother the attendant a great deal to know the best method of treating such a case; it must be cured, else it would result in some very serious conditions.

I might mention as incidental and pertinent to this subject thread-worms in the infant; they often occur in adults as well, and are very distressing, though at first sight they would appear to be exceedingly simple; it often bothers one not a little to know how to get rid of these conditions.

I might go on and mention a number of diseases of the infant that we have in a rectal way; then between the ages of eight and ten up to twenty years there are some special diseases of the rectum that will be found both in the male and female, no one being exempt, in which, as I have previously indicated, one may never be able to trace the cause.

All this being true, I would emphasize the necessity of one thing, *i.e.*, examine the patient thoroughly. It is surprising to know, even when the patient calls attention to slight trouble about the rectum, how few doctors ever examine the rectum to see what is the cause. And, therefore, very grave mistakes are made.

A few days ago I went to the country and operated upon a young man who had visited me the day previously. He gave this history: That there was a swelling in one buttock; but he did not seem to think it amounted to anything, because he said that it had never "pained him." I said to him, "I am sorry, my young friend, that it has never pained you. I would be much better pleased if you would say that it had been exceedingly painful." If there is a swelling, and this swelling contains fluid, if it has not given him any pain, it is nearly a pathognomonic symptom that the man has what? Tuberculosis; that the condition is tuberculous; because he has had no pain leads his physician to say to him, "It cannot amount to anything," and leads the patient to think that it amounts to but little; then, when I operate upon the young man, I find the whole left buttock actually destroyed by a tuberculous process. Therefore it is necessary to carefully examine people who complain in the least of rectal trouble; simply because they say, "Doctor, it is not necessary, it does not hurt me," do not let that have any significance, but make a thorough and painstaking examination.

The physician does not need to have a great number or an extensive set of instruments to examine people for diseases of the rectum; not once in fifty times will he need to use a speculum, at least, he should not; it reveals but little, and the most reliable information that can be secured is by the introduction of the examining finger. The mucous membrane in the normal rectum is not like mucous membrane anywhere else; it is a thick membrane, movable, and it is different in color from mucous membrane in other situations.

The surgeon should never do an operation upon a fistulous tract until he has introduced his finger into the rectum to see if there is any possible complication, because above the fistula might be a stricture, cancer, or some other condition. Having anointed his finger with vaseline, it should be introduced into the rectum; and he will often find that there are warty excrescences upon the mucous membrane extending throughout the entire circumference of the gut, the nature of which may at the time be hard to explain. The sphincter

muscle may be very feeble. Now, in such a case, although there may be a number of fistulous tracts, the surgeon should not take the trouble to find them; introducing a probe into the external opening, he may find it enters a cavity from which he will get his bearings as to other fistulous tracts. He may find an area as large as his hand involved, extending two and a half inches or more out into the buttock. There may be an elevated condition of the skin itself, nodules, etc., having the appearance of syphilis; whereas, they may not be true condylomata; they may assume that nature, and fistulæ, of course, may result from a diathesis of that kind. I may say there is always a suspicion of syphilis in colored people, whether it can be verified or not. Allingham once said to me that he had seen dozens of cases where the only syphilitic manifestation was in the rectum, and my experience has been the same.

Cases of this kind are exceedingly disagreeable to operate upon, the condition is very ugly; and it is necessary to use some means of protection against infection from the local condition, which may be done by thoroughly anointing the hands with vaseline. To do such a patient as I have described any good, at least a portion of the buttock would have to be removed. No special rule can be observed in operating upon cases of this kind, so far as operation for the fistula is concerned, because it is so complicated as to be a very serious condition.

I have always tried to impress upon students, in their observation of these unfortunate people in the clinic-room, that the same conditions may be met in private practice.

In operating upon cases such as I have mentioned, instead of introducing a grooved director and opening the sinus in this way, it is better to simply remove a portion of the buttock by dissection; of course, all the sinuses are included in this dissection. In making a dissection of this kind, quite a good-sized artery may sometimes be severed. This might be clamped; but I prefer to tie it, as that part of the integument might be drawn up into the rectum and secondary hemorrhage might result, and later give trouble. The section removed will be found to consist simply of a mass of necrosed tissue, with sinuses running through it in different directions. If other sinuses are found running back towards the rectum, it will be necessary to remove more tissue.

Underneath the tissues removed by dissection there are often

other sinuses, or an extension of those observed running out in various directions, some going down deep into the tissues. These should be curetted, care being taken to get to the bottom. In any direction the probe is introduced, additional sinuses may be detected, for which, being too deep for the curette to be of service, the surgeon should again use the knife and scissors. After removing more of the tissue he will then curette down to a healthy base, and trim off more of the edges, if necessary. Drainage in such a case is very important, whether it be syphilitic or whether it be tuberculous. The patient may take chloroform badly, and cannot be kept thoroughly under its influence, therefore the operation is rendered exceedingly difficult.

In a recent operation of this kind, I observed a sinus extending towards the vagina, into which I could introduce the probe, and the patient would never have been cured of the condition unless I had also divided that. After dividing this sinus I left two edges, which also had to be trimmed off. After getting down to good tissue everywhere, excepting the point where the sinus led to the vagina, I found another indurated spot just at the edge of the anus which would never have healed, therefore it was cut away. Another sinus was detected running two and a half or three inches out into the buttock, which was divided, and the edges trimmed off as was done in the others.

These patients are kept in bed for a short time; in four or five days they can get up and walk around the ward, and the wound will drain just as well, if not better, when in the upright position. It is often a mistake to keep patients who have been operated upon for fistulæ in bed for a long time. The wounds will heal just as well if they are allowed to walk about, and the advantage is that they will gain in flesh and strength, the appetite will improve, and the secretions of the body will likewise be improved.

It used to be presumed that all fistulæ were tuberculous in character, and that to operate upon the fistula would cause premature death of the patient. Of course, that theory has long since been exploded. It is true, however, that there are a great many fistulæ the result of a tuberculous diathesis; and even in these patients operation should be done promptly, and the patients should be allowed shortly afterwards to get out in the open air and take plenty of exercise, and under those conditions I believe such cases are curable.

This raises the question, which is very interesting and one about

which there has been considerable discussion, *i.e.*, the supposition that because, forsooth, a man has a fistula it has a direct connection in some way with the lung. The profession in the past, and even some of them at the present day, state that there is such sympathy between the fistula and the lung that it would not be advisable to cure the fistula. They take the position that if the fistulous tract heals that phthisis will be established, or, if phthisis was already established, then it would extend with great rapidity, and the patient would die in a short time. They even went so far in the past as to say that if a person had a fistula, and you cured that fistula, it would produce consumption.

How erroneous these views are can be readily understood, because, since the discovery of the tubercle bacillus, the fact is recognized that a person may have tuberculosis anywhere about the body from the scalp to the soles of the feet. Sometimes it affects the scalp, frequently the rectum, the joints, etc.; and as far as treatment is concerned it is exactly the same that you should employ in the attempted cure of cancer,—there is a local focus of infection which must be removed in order to effect a cure. This is the reason I said to the young man already referred to, operated upon in the country, "It is fortunate, even now, that you have had the operation performed, because, if any of the tubercle bacilli were left, you might have general infection." Applying these remarks to other cases, there is a probability that by this operation we will save the local distribution of pus, but more than that, we may save the patient, presuming that the condition is tuberculous, from general tuberculosis. A simple incision should not be made; the various channels, sinuses, etc., should be thoroughly laid open, and the surgeon should trim and curette away every single portion of tissue that is infected. By the free drainage that will be afforded, and the antiseptic treatment that the wound will receive, in all probability the patient will be prevented from having general tuberculosis.

If a patient, even if he be a tuberculous subject or has consumption, if you please, having a fistula that is giving trouble, applies to the surgeon, he should not hesitate to operate; or, if the patient is seemingly predisposed to consumption and has a fistula the surgeon should operate. Indeed, I can conceive of no condition of fistula that should not be operated upon except one, and that is where the rectum is so blocked, for instance, with syphilitic or cancerous deposit

that the fistula is simply secondary to this condition caused by ulceration above, and, consequently, a cure could not be obtained, but the distress of the patient would be aggravated by laying open the fistulous tracts. That is the only instance I can call to mind where an operation for a fistula proper is not justifiable. Certainly, it is advisable to operate upon a consumptive patient for fistula.

BONE LESIONS OF HEREDITARY SYPHILIS IN CHILDREN.¹

CLINICAL LECTURE DELIVERED AT THE CHILDREN'S HOSPITAL, BERLIN.

BY PROFESSOR ADOLF BAGINSKY,

Director of the Kaiser und Kaiserin Friedrich Kinderspital (Children's Hospital),
Berlin, and Professor of Diseases of Children at the University of Berlin,
Germany.

GENTLEMEN,—I have here this morning two characteristic cases of a very important disease, with which you will often be brought in contact if you have—as you all will, of course, later on in life—a large practice in children's diseases. They are characteristic of certain symptoms of the disease which are in themselves typical, though not its most frequent manifestation. The affection is hereditary syphilis, and its immediate manifestations in both these cases are bone lesions; in one in the very early acute stage; in the other, in the late chronic stage, one might almost say the post-syphilitic stage.

As I take off the coverlets from this nursling of five weeks, you will note that it is sleeping quietly, looks chubby, rosy, and well nourished, and might be thought perfectly healthy except for its attitude in the crib. A nursling's arms should always be found, especially during the first few months of life, bent at the elbows, with the hands up along-side the head. Here they are lying along-side the body, and if you look at them closely, you will see that they do not seem to be merely lying unconstrainedly in that position, but that there is an appearance of constraint in their almost complete extension, as if the child was forced to hold them so from dread of pain.

When a nursling's arms lie along-side its body, as here, there is always something wrong, and if but one of them is down, the other being held in its usual position, the lesion is a one-sided one. This may be the earliest symptom to call attention to some of the possible accidents of parturition. If the humerus has been broken by some

¹ Reported by James J. Walsh, Ph.D., M.D.

manipulation during the delivery, or if there has been a dislocation of the shoulder or elbow or a fracture of the clavicle, then the pain of movement will cause the child to let the arm lie extended along its body; otherwise it would surely lift it upward, as it has become accustomed to that position during intrauterine life, and the development of the muscles in that position originally naturally causes a reversion to it during the unconsciousness of sleep, until in the course of time new muscular habits are learned.

Of course, this allowing the arms to hang downward may mean paralysis too, commonly one of the forms of birth palsy due to injury of the brachial plexus, when usually the lesion will be but one-sided. There may be paralysis of both arms, however, due to double cortical hemorrhage during birth. In our case here, even before we touch the arms, I think we can see that there is no palsy present, the constrained position of the arms being very different from the relaxed, helpless-looking way they hang when paralysis is present.

There is a pseudoparalysis, but it does not require much to demonstrate beyond a doubt that it is not true paralysis that is in question. The moment I lift this arm, though gently, the child starts from its sleep with a cry of pain. Here, just above the elbow in the right arm, there is a distinct swelling, and it is evidently here that the principal seat of pain is situated, from the way the child complains when we touch it.

Now, there is no history of trauma in this case; the symptoms have developed in the last few days, so there is no question of an accident of parturition, since the child was well for a month. Besides, the position of the swelling, just at the point where the diaphysis of the humerus meets the epiphysis, is characteristic of other affections rather than of a fracture at this age. We look for some symptoms to confirm our suspicion of a syphilitic epiphysitis, and we find them in these few slightly raised papules covered with a few whitish scales on the foot-soles. The cutaneous lesions would scarcely be noticed unless especially looked for, but in this location they are characteristic, and we have a slight but typical syphilitic psoriasis.

We have, then, in this case a good example of what has been called syphilitic osteochondritis, or what Wagner called syphilitic osteitis. I have often been surprised that surgeons did not lay more stress on these syphilitic manifestations in the long bones of children affected by hereditary syphilis, for I have found them to occur rather

frequently. In my experience, contrary to what Parrot has reported, I have found the arms most frequently affected, though the difference in frequency of the affection in the arms and in the legs was not great. In our case the legs are free. There is, however, a suspicious enlargement here above the elbow of the left arm, and seemingly a tenderness to the touch that is at least suggestive of a corresponding affection in the other arm. It is, too, as we noted in the beginning, held in this pseudoparalytic position.

Attention has often been called to the fact, however, that this pseudoparalysis syphilitica of Parrot might at times be a genuine palsy due to a nervous affection. Henoch believed that at times the peripheral nerves were affected as well as the bones, or that the symptoms in certain cases were not due to a bone affection at all, but directly to a syphilitic neuritis.

Quite recently Zappert, working in Obersteiner's laboratory in Vienna, has been able to demonstrate pathological changes in the cord in a case of supposed pseudoparalysis syphilitica. The case was presented as such before the Vienna medical society, and the diagnosis was made of syphilitic epiphysitis. The child had a series of extremely severe syphilitic lesions,—iridocyclitis and interstitial keratitis, etc.,—and died two weeks after the presentation before the society. No changes were found in the bones, and so the brain and spinal cord were carefully prepared by Marchi's method. The arms had been mainly affected, and in the cervical enlargement was found a series of pathological changes, beginning with a meningitis. The pia was thickened and contracted at the point where the nerve-trunks pass through it to the cord and where they are especially sensitive to pressure,—in a word, at the point where Redlich and Obersteiner have pointed out that a syphilitic sclerosis and contraction of the pia would give the degeneration in the posterior columns of the cord which constitutes the characteristic lesion of tabes dorsalis.

The case is a most interesting and suggestive one; interesting because the first one in which definite lesions have been found in nervous tissues, though the acute observation of Henoch led him to suspect them before the technical methods for the examination of nervous tissue were sufficiently perfected to demonstrate them. On the other hand, it is as yet an isolated example, and as such must be accepted with some reservation as a typical case. The original observations of Parrot in syphilitic epiphysitis were substantiated by

autopsy, and many others have been since, so that the type of Parrot's disease remains. Whether we shall have to admit a pure paralysis syphilitica at times, with a genuine syphilitic nervous lesion alone as its cause, remains to be seen.

Conclusions drawn from the appearances of degeneration in the nervous tissue of children must always be accepted guardedly. The process of development is still in progress at birth and continues for months afterwards. Differences from the normal nervous tissue of adults or older children may easily be found and considered pathological, when they are only the manifestations of imperfect development. We do not as yet know enough about the normal microscopic appearance of children's nervous tissue at early ages to draw any sure conclusions from supposedly abnormal appearances. If we needed a warning in this matter, the division of opinion some years ago between Virchow and Jastrowitz would act as one.

Virchow found in certain cases in very young children fat-globules in abundance in certain parts of the cerebrum. This fatty metamorphosis could not be physiological, and was not the expression of incomplete embryological development; so he considered it due to encephalitic processes. Jastrowitz announced some time afterwards that he had found exactly the same appearances in perfectly normal children.

Certainly in most of these cases of pseudoparalysis syphilitica, if nerve lesions exist, they are of a character which allows of complete restoration to the normal, with perfect recovery of physiological function, for nearly all these cases completely recover the use of their limbs, and where lowered function remains after the attack there is usually some bone lesion to account for it. That one should find degenerations with the Marchi method means a permanent injury to nerve-trunks and tracts, which must occur only in extreme cases, such as the Vienna case is reported to have been, and which usually, therefore, run a fatal course. I should consider that here we have a case of syphilitic epiphysitis, and that the paralytic symptoms are secondary rather than primary.

As to the prognosis in a case like this, it is not unfavorable either as regards life or the recovery of function in the limbs. In general the prognosis of hereditary syphilis depends on the condition of the little patient. Thin, delicate, miserable-looking children, so from birth, whose general nutrition is distinctly lowered from the begin-

ning, whose vital resistance is diminished because their proper share of vital nerve-force was never apportioned to them, succumb readily when the severer symptoms of hereditary syphilis begin to develop. Children that are chubby and healthy-looking when born, and whose parents are of good natural constitution though infected, one or both, with syphilis, struggle through the symptoms of hereditary syphilis very successfully.

Our child here looks rugged and healthy, despite its syphilitic taint, and I have not the slightest doubt will pull through all right. As to the prognosis of the recovery of function in the affected limbs after pseudoparalysis syphilitica, that too is very favorable. Of course, it depends entirely on the extent to which the inflammation of the epiphysis goes. Usually there is syphilitic neoplastic growth in and around the epiphyseal cartilage, a true osteochondritis syphilitica. In an early stage this is represented only by a hyperæmia of the marrow spaces in the vicinity, and, as this occurs also in rachitis, too much value should not be attached to it post-mortem as being pathognomonic of syphilis.

Later in syphilis the blood-supply is interfered with by the syphilitic endarteritis, and a fatty degeneration, sometimes with the development of gummatous nodules, takes place. Meantime the irritation has led to new tissue-formation, and the ends of the bones, especially near the epiphyses, become thickened, though sometimes the whole bone becomes thickened. If the process can be stopped at this point by specific medication, the recovery of function is assured, as a complete absorption of the syphilitic products and involution of the syphilitic process will take place, although you may have the pain and tenderness and the pseudoparalysis as we have it in this case, and though they may persist for some time.

Where the process goes further, and either spontaneously or because of some slight injury the epiphysis becomes separated, the prognosis as to recovery of function of the limb and of the bones developing properly in length, since it is on the epiphysis that this depends, is much more doubtful. In these cases gentle manipulation will give crepitus, though one must be careful not to mistake for this crepitus, which is much less rough and grating than that usually discovered in a fracture, because the bones here are soft and one of the parts is cartilaginous, the creaking crepitant sensations sometimes developed in the dried tissues of the joints of marantic children.

Even where separation of the epiphysis has taken place, there will usually be under proper treatment a union of diaphysis and epiphysis once more, and function and development will remain undisturbed.

Where the epiphysitis goes still further and becomes purulent, of course the prognosis as to restoration of function is very unfavorable, and deformity and maldevelopment are almost inevitable. At times the epiphyseal cartilage or portions of it are thrown off as a sequestrum, and it is wonderful that, even under these unfavorable circumstances, nature's recuperative efforts are sometimes sufficient to restore parts or so compensate for them that not all the deformity which might be expected occurs, but a useful, not over-dwarfed, limb remains for later life.

In this case the syphilitic epiphysitis, except for a slight attack of snuffles that had been unsuspected, was the first notable symptom of the hereditary syphilis observed. Usually other and what are considered more typical symptoms have developed before it and given warning of what the nature of the process is. Here after careful search we have found the spots of syphilitic psoriasis on the foot-soles, and there is about the skin of both palms and soles a varnished look that would make one think of syphilis. If you pay attention you will note that the child snuffles slightly and that it breathes mostly through its mouth; there is also a harsh crowing-like sound about the cry that is suspicious. These have become noticeable during the last day or two.

Here around the anus there is nothing suspicious, though this is usually where the first cutaneous symptoms are seen. A good deal depends, however, in the occurrence of cutaneous syphilitic lesions, on the amount of irritation that parts are subjected to. Men who wear hats that produce irritation on the forehead and at the margin of the hair have the corona veneris. Women who wear collars that irritate are more troubled by the collar of Venus. In children where cleanliness is neglected the manifestations of the disease will be more outspoken on the genitals and the anal region; if kept thoroughly clean there is not much more liability to syphilitic eruptions here than in other parts. This is a practical point in the prophylaxis and treatment of cutaneous syphilitic lesions in children that has not been sufficiently insisted on.

Many methods of giving mercury to children affected by hereditary syphilis have been suggested. I think that I have faithfully

tried them all, and consider that the method which employs mercurial baths is the best and most effective, and besides has associated with it the least inconveniences. The baths are given about as follows: for children under one year of age half a gramme to a gramme (eight to fifteen grains) of bichloride of mercury are dissolved in twenty to twenty-five litres (about twenty quarts) of water, and the child is bathed in the solution for about half an hour once a day. These baths are continued daily for about twenty-five days, with the exception of every eighth or tenth day, when there is an intermission for a day.

Usually at the end of this time there has come a suppression of symptoms, and the baths may be omitted for ten days to two weeks and then begun again. After the second series of baths treatment may be interrupted for a time until further symptoms are manifest. Of course I need scarcely warn you that mercurial baths should be given in wooden tubes. Most of the metals are acted upon by mercury and lead to its precipitation, or rather to the formation of an amalgam. This distinctly weakens the mercurial solution, besides leading eventually to the ruin of any metal vessels that may be employed.

From these mercurial baths for nurslings I have always gotten excellent results. I have employed them for a good while now, practically to the exclusion of every other means of medication, and I have been perfectly satisfied with the results I have secured. In fact, I have almost never had a case where they have failed me. I have found them especially effective in the severe osseous or osteochondritic syphilitic affections in pseudoparalysis, as here. The pain and palsy usually completely disappear in five days, though the syphilitic neoplastic process takes a little longer than this for its complete involution.

I prefer the mercurial baths to internal medication or to the rubbing of mercury into the skin. Taken internally mercury disturbs the digestion, and it is on its power of absorbing nutriment properly that the infant's life depends in the siege of syphilitic invasion that is before it. For mercurial inunctions the infant's skin is too tender and too easily irritated, though if the baths did not succeed this would be the form in which I should employ the mercury, in order to spare the digestive apparatus. As to mercurial injections, I do not consider them feasible in small children, because of the irritation they are liable to set up.

In whatever form the mercury be employed, it must be remembered that nurslings, even though they have no teeth and secrete no saliva, are still liable to the mercurial mouth lesions which accompany salivation without the dribbling. I have seen quite bad ulcerative mercurial stomatitis develop in cases where this fact seemed to have been forgotten. In all cases the infant's mouth should be carefully washed out with a boric acid solution, very gently, of course, for the slightest roughness that abrades the delicate mucous membrane may give a foothold for the beginning of the stomatitis. Where stomatitis is present, washing with dilute solutions of chlorate of potassium will do good; but it must be remembered that the potassium chlorate is itself an irritant poison; consequently much of it should not be used, and the mouth should be washed out afterwards with plain water.

Even when there are eczematous or ulcerative skin eruptions the baths are well borne, the eruptions are not irritated and made worse, and not too much mercury is absorbed. For local syphilitic lesions I employ local medication. Condyloma latum around the anus or on the genitals I touch with the following solution:

R Hydrargyri chloridi corrosivi, gr. iii;
Spiritus vini,
Aqua destillatae, aa $\frac{3}{4}$ i.

Even with the bath treatment, there will occasionally be relapse of symptoms, and these should be carefully watched for, so as to anticipate any serious mischief. Especially should the eyes be carefully looked after, as here serious damage may accrue most insidiously.

For purulent and other arthritic affections surgical rules must apply. Where separation of the epiphyseal cartilage has taken place a splint should be applied; where a collection of pus exists it should be evacuated.

Not all the children that have had symptoms of hereditary syphilis develop rachitis, but a great many of them do so. In all these cases the first symptoms of the rachitic process should be carefully looked for and met with proper remedies. The best possible prophylaxis against rachitis, however, is proper nutrition of the child. This is, of course, supplied best by nursing, and when for any reason this is impossible, the prognosis of the case is distinctly worse. I do not think, however, that we are ever justified in allowing a healthy

wet-nurse to suckle a syphilitic child, even when the nature of the case has been properly explained to her and she is nevertheless ready for a consideration to take the risk. No proper understanding of the seriousness of syphilitic infection can be given these people, and the danger of further spread of the disease is too great.

It must be remembered that children in the early stages of hereditary syphilis can readily communicate the disease to others. This is sometimes forgotten, and other children born before the syphilitic infection of father or mother may be infected. Other children and friends of the family should by no means be allowed to handle or caress or kiss the baby or use anything meant for the baby's use. I have seen a number of cases of contagion from hereditary syphilis, and so here at the hospital the syphilitic children are always segregated from the other children in a room by themselves.

At present there is here, as you see, only one other, a girl of about twelve, whose case is interesting as showing the stigmata of hereditary syphilis after the acute symptoms of the early years have disappeared. She is not in the hospital because of any syphilitic manifestations, but for a pericarditis, perhaps of rheumatic origin, so that we shall endeavor to disturb her as little as possible during our examination. She has not the outspoken look of hereditary syphilis,—*i.e.*, there is no saddle-shaped, broad, sunken nose, and she has neither the look of precocious senility so characteristic of most of these cases nor the look of extreme youth for her years,—the infantilism, as it has been called, due to syphilitic paediatropy. She shows her age pretty well; she is past twelve, and she is of good size and normally developed.

There are, however, certain other unmistakable signs that point to the fact that she has suffered from a series of hereditary syphilitic lesions. Her eyes indicate that there has been a chronic inflammatory process at work; her cornea lacks the glance of the perfectly normal cornea and betrays the interstitial keratitis that has been present. At the angles of her mouth there are white radiating scars, the marks of healed rhagades or syphilitic fissures, whose favorite locations are where skin and mucous membrane meet, especially where an angle occurs, as at the corners of the mouth or in the centre at the normal fissure of the lip.

When we have her open her mouth her teeth at once attract attention. They are irregular, naturally; they are abnormally carious

for such an age; they are imperfectly developed, and some of them have the appearance of pegs stuck into the gums,—the “pegged teeth” so often described. The two central incisors are a characteristic pair of so-called Hutchison teeth, the distinguished English surgeon, Jonathan Hutchison, having considered them pathognomonic of hereditary syphilis. The external surfaces of these teeth are comparatively normal, but the cutting edge and the inner surface are decayed. There is in each an almost crescent-shaped defect from caries, and the inner surface has a honeycombed porous appearance. The teeth are not on the same straight line, but are turned towards each other internally, which is more commonly the case than the external rotation away from each other, though this is also seen at times.

As to these changes in the teeth being pathognomonic of syphilis, I do not think them so. I think that such changes may occur with any serious disturbance of nutrition. We have here two of the Hutchison triad of symptoms of hereditary syphilis,—the remains of the keratitis and the deformed teeth; but we have no deafness and no history of any having existed. This auditory symptom is the one which I think is oftenest missing of this group of three. We do not, however, in our present case need the “triad” to confirm the diagnosis, as there are enough other symptoms. If you look into the mouth you will notice the white radiating scars on the soft palate and even in the back wall of the pharynx, which can only be post-syphilitic. Besides, see how thick and rough and bowed anteriorly her tibiae are, and how much harder and more porous they seem on palpation and percussion. Here you could not miss the signs of the syphilis that has existed, as is so often the case they are indelibly and unmistakably imprinted on the tissues of the patient.

Where there are no active symptoms of the disease, as in this case, no treatment is required. If symptoms were manifest I would give the iodides,—not sodium iodide nor potassium iodide; they are too unpleasant for children and disturb their appetites seriously, but the saccharated iodide of iron in doses of from one to five grains three times a day. This is a pleasant and effective preparation of the iodides, and has the advantage, too, of being useful for the anaemia that so often accompanies even the late manifestations of syphilis, and constitutes a very prominent indication for treatment in most of these cases.

TECHNIQUE OF HÆMOSTASIS IN OPERATIVE LESIONS OF THE LARGE VEINS.

CLINICAL LECTURE DELIVERED AT TUFT'S COLLEGE MEDICAL SCHOOL.

BY CHARLES GREENE CUMSTON, B.M.S., M.D.,

Assistant Professor of Surgical Pathology, Faculty of Medicine, Tuft's College, Boston; Honorary Member of the Surgical Society of Belgium; Fellow of the American Association of Obstetricians and Gynaecologists; Corresponding Member of the Association of Genito-Urinary Surgeons of France, of the Obstetrical and Gynaecological Society of Paris, of the Pathological Society of Brussels, etc.

GENTLEMEN,—You will probably be somewhat surprised when I tell you that I am to refer to-day to the small boy that was operated upon yesterday, and whom you saw at my clinic a few days ago. I should not have thought of the case again had it not been for an accident that occurred during the operation.

The history of the case is in brief as follows: Fred. J., aged twelve years, applied for treatment for a small tumor, about the size of a large walnut, in the right submaxillary region. The tumor was rather painful, somewhat lobulated, and the skin covering it was freely movable and otherwise normal.

The patient had never been ill, and his parents and two brothers are alive and in good health. The neoplasm was first noticed by his mother about five months ago, and has continued to increase in size ever since. A diagnosis of lymphoma of the neck was made and extirpation advised.

Yesterday we operated, and while trying to dissect out the growth the internal jugular vein was cut longitudinally for about seven centimetres. The wound in the vein was at once plugged with gauze, and then with a round needle and fine catgut the opening in the vessel was closed by a running suture, and the sheath of the vein was next brought together over it with a few stitches. The neoplasm was then removed, a gauze wick was inserted for drainage, and the skin incision was closed with silk.

The dressings were removed to-day. The wound was in fine con-

dition, and by to-morrow I think we can do away with the drainage entirely.

One of the most unpleasant things that could happen to a surgeon in the preantiseptic days was an injury to a large vein during an operation. Not only was this mishap dangerous on account of imperfect means of hæmostasis, but on account of the unfortunate consequences, such as entrance of air into the vein resulting in death, secondary hemorrhage, phlebitis, pyæmia, thrombosis, and gangrene. Many of these complications simply arose from the entrance of pyogenic bacteria into the vessel where they rapidly proliferated on the excellent media offered them in the form of blood-clots. A phlebitis was thus set up which rapidly extended; a thrombosis took place, the thrombus suppurred, and bits of it were soon carried throughout the blood-current, and pyæmia was the natural consequence.

As you see, it has been a most important point for all practical surgeons to find a method which would safely occlude any opening made in a vessel during an operation. Many were devised, and all were condemned as dangerous, because septic complications arose; but it was not the means employed for hæmostasis that were at fault, but the want of asepsis, causing the patient's death from infection.

But with our present perfected surgical technique and aseptic principles, a large number of these complications are done away with, and phlebitis and pyæmia are now rarely seen, and if by chance they should occur we are better prepared to combat them with many therapeutical measures that modern medical and biological science have placed at our disposal. Thrombosis and the resulting gangrene are practically unheard of to-day after a lesion to a vein, and the risk of the entrance of air into the vessel is about the greatest danger to be feared. This will occur in those instances where, after the accident has taken place, the vein does not collapse, so that the walls remain apart and with each breath a valve-like action takes place, aspirating air directly into the lumen.

This most frequently is the case with the large veins near the heart which are directly influenced by respiration, such as the sub-clavian vein and internal jugular, or even the external jugular; when in unusual circumstances they are prevented from collapsing, air will rapidly rush in, and for this reason you should always be prepared to act in this emergency when operating about the neck.

A single or several separated air-bubbles are not quite so dangerous in their effects, as they are gradually expelled from the blood.

As to the danger of secondary hemorrhage, it may be said that it depends entirely upon the manner in which the vein has been closed. At the present time we ligate freely, and large venous trunks are resected. A lateral cut or tear of a vein of some size is always to be sutured when possible, because a total ligature is sometimes followed by unfortunate complications, an incomplete collateral circulation being established.

The principal venous trunks that you should bear in mind in this respect are the jugulars, the axillary, and the femoral. Of recent times much discussion has arisen over the best method of haemostasis in lateral lesions of the veins which will not disturb the function of the vessel nor interfere with union per primam of the wound.

Now, a lateral opening in a large vein,—in this lecture I only refer to the large trunks, as small ones are simply ligated and cut,—may occur under most varied circumstances, but the larger number of cases are certainly due to an accidental nick of the knife during extirpation of neoplasms situated in the neighborhood of or bound to large veins, and when the vessel is intimately adherent to the growth, it is often not recognized until opened. But since the use of surgical anæsthesia wounds of the veins during operation are far less frequent than formerly, because we can work slowly and deliberately; nevertheless, this mishap will happen, even when great care is taken, when the vein is not filled with blood and may be mistaken for a cord of connective tissue, as happened to Von Langenbeck.

When removing neoplasms the non-adherent veins can be easily held aside, but they may slip away without notice, or they may be abnormally situated and the surgeon comes upon them unexpectedly. Traumatism may also produce a lateral opening in a vein. Then, again, a vein may be so intimately adherent to a neoplasm that a portion of it must be resected along with the growth, but this is done by first ligating the vessel above and below before cutting it, or, if it is an important trunk, we might, as has been done by Schede, empty it of its blood by pressure and, after securing the vein above and below the tumor, resect that part of the wall adherent to the growth, and after this has been extirpated suture the vessel together.

The principal methods now in vogue for treating lateral openings in veins so as to preserve the lumen are as follows: 1, compression

by tampons or plugging; 2, lateral ligature; 3, temporary clamping (clamps à démeur of the French); and 4, suture of the opening. I will now consider separately each one of these methods, and will begin with that of plugging with compression.

Compression is the original, and in olden times the most common, method of procuring hæmostasis. The opening is plugged with gauze or gauze sponges, and lateral pressure is brought to bear upon the lesion to prevent the access of air and to promote a coagulation of blood which will obstruct the opening. Compression was done with either the fingers or with certain apparatus, but sepsis usually resulted from direct infection.

At the present time aseptic or antiseptic tamponing has been practised in small openings in the veins, the compression producing the formation of an intra- and extra-vascular coagulation of blood, which later will be converted into tissue of new formation.

Maubrac has reported a case of a gun-shot wound of the femoral vein in which plugging with iodoform cotton resulted favorably. The patient died, however, on the third day after the injury, death being due to a septic peritonitis produced by a shot in the abdomen, received at the same time as the bullet in the thigh. At the necropsy the femoral vein, which was directly penetrated by the ball, had begun to undergo the process of repair, and the surrounding tissues did not present any sign of extravasation. No thrombus was present in the lumen of the vessel, which proved that the vein remained patent.

In more extensive injuries, however, it is most probable that the vein is displaced from the compression exercised, so that circulation will be interrupted and the natural result of the plugging will be much the same as in the case of double ligature. Tamponing can consequently only be successful in those cases where a collateral circulation takes place rapidly, a thing that must rarely occur when we are dealing with a large vein which is the chief vessel of the region it supplies.

Kay believes that plugging belongs to the methods which obstruct the lumen of the vein, and only recommends it in cases where all other measures fail.

With plugging and compression it is often difficult to obtain a complete suppression of the circulation through the vessel in certain regions, as, for example, in the neck; but in the limbs it gives less

trouble. When the opening in the vein is longitudinal the limb should be put in complete extension; when the wound is perpendicular to the axis of the vessel the limb should be flexed so as to bring the edges of the opening into the closest approximation possible. Tamponing and compression are most useful, and are particularly indicated in openings of veins from erosion or when a sinus has been opened during operations on the brain or mastoid process.

We now come to the second method of closing wounds of the veins,—namely, lateral ligature, an operation first performed in 1816 by Travers, according to Blasius. Travers's case was an injury to the femoral vein, which he tied by a lateral ligature. On the tenth day the ligature came off, and on the following day a secondary hemorrhage began which could not be controlled, and the patient died on the thirty-third day following the injury. Autopsy showed that the tunics of the vessel walls were thickened, the lumen was narrowed more and more as it neared the wound, at which point it was completely occluded. At about three-quarters of an inch above the occluded portion a small opening was found. An adhesive inflammation had taken place, beginning on the inner surface of the femoral vein and extending as far as the point where the iliac vein enters into the inferior cava. The latter was also the seat of an inflammatory process, but all trace of suppuration was absent.

In 1822 Von Wattmann reported a successful result of lateral ligature of the internal jugular vein; on the twelfth day the ligature came away, and in about a week after the wound was entirely closed. The same surgeon had another case of lateral ligature of the internal jugular, but death resulted from pneumonia on the twentieth day.

Since this time lateral ligature has been more frequently resorted to, and was very much praised, but soon again was condemned. The most fatal blow to this method was given by Blasius, who greatly condemned the method and helped to place lateral ligature in dispute. In his work on the subject Blasius cited sixteen cases of lateral ligature of veins, only seven of which did he consider as proper for the operation; 71.5 per cent. resulted fatally, and only 28.5 per cent. were successful, the success only being due to a thrombosis.

This authority undertook fifty-six experiments on rabbits, with a successful result in but 37.9 per cent. of instances, and in these there occurred a thrombosis, and in no one instance was the lumen of the vessel patent. After Blasius's monograph appeared lateral ligation

of the veins was practically abandoned until 1882, when H. Braun took the question up again. Of the nine cases that Blasius excluded as unfit for lateral ligature, Braun considered four of them as unsuitable, but in the remaining five he believed the operation was justifiable. Now, of the twelve cases considered by Braun as proper for lateral ligature, five ended fatally and seven recovered, thus making a mortality of 41.6 per cent.

Braun also cited seventeen new cases, ten of which were successful. Two patients died so soon that death could not be attributed to the ligature, while of the remaining five fatal cases, only two could be considered as resulting directly from lateral ligature, because the three others died from pyæmia, a complication that would have taken place if the vein had not been tied. All told, there are twenty-seven cases recorded by Braun, twenty-four of lateral ligature and three by clamps à démeur, with a mortality of 38 per cent.

Blasius believed that the greatest disadvantage of lateral ligature was the fact that the vessel was always occluded by thrombosis, so that the ultimate result is the same as total ligature, and he recommended the latter because it exposed the patient to fewer dangers, such as slipping off of the ligature and secondary hemorrhage following its coming away.

At the present time we cannot uphold the formation of thrombosis as an objection to this method, because antiseptic technique has done away with all danger, and well-conducted experiments have proved that the operation is devoid of danger.

Braun experimented on rabbits with lateral ligature, and demonstrated that the lumen of the vein remained patent, and in one animal examined nine months after the lumen was perfectly preserved at the point where the ligature had been applied.

We may consequently conclude that a carefully applied lateral ligature will not occlude the lumen of the vein, but naturally its caliber will be diminished at the point of ligation, which will not seriously influence the circulation through it.

But there is a more serious objection which in olden days was more of a complication than it is at present. I refer to the dropping off of the ligature which takes place at about the eighth or tenth day, and which was often followed by a secondary hemorrhage; but at present an antiseptic silk or catgut ligature will not slough off, but will become encysted or absorbed according to the material employed.

Still another objection, which has by far more weight even now, is the fact that, the walls of a blood-vessel being very smooth, the ligature gets no hold and may slip off, with the result that we get a primary hemorrhage.

Slipping off of the ligature is not necessarily due to the smooth surface of the vessel, but because of traction produced by the inevitable shortening of the vein from the application of a lateral ligature. After the ligature is applied a cone is formed in the vessel walls with its apex pointing outward, and if the ligature gets loose it naturally easily slips off.

Now, if the opening in the vessel has not become occluded by the process of repair, and this cannot be expected to take place in less than twenty-four hours after the operation, a secondary hemorrhage is sure to occur if the ligature slips.

Braun demonstrated that in the cadaver a lateral ligature would stand a pressure of from seventy to one hundred and eighty millimetres of mercury without slipping, but whether this can be done in the living, where we have in addition the elastic traction and a smoother surface, is not as yet known.

Practically, then, we may say that lateral ligature is to be only employed in small openings in the veins, not exceeding four or five millimetres in length. It is quite impossible to close a wound of a vessel which is larger than this without interfering with the calibre of its lumen. Braun states that if not more than one-third of the circumference of the vessel be injured a successful result may be obtained by lateral ligature.

Lateral clamping of the opening in a vein will now be considered. In the '60's Porter and Pirogoff recommended the use of artery forceps left *in situ* for from twelve to twenty-four hours in place of lateral ligature, although this method had been recommended by Von Wattmann in 1823 and by other surgeons long before him.

In 1887 Schmidt described the use of clamps à démeur, and records a long series of experiments on animals. This surgeon used rabbits, and experimented on the jugular and femoral veins. The vessel was opened with either scissors or knife, and the wound was brought together with several modified serrefines. The cutaneous incision was closed with silk and covered with iodoform gauze. In twenty-four hours the dressings were removed, and the serrefines were taken out with great care and the line of incision closed with

collodion. In from six days to two weeks the vein operated on was resected for examination.

The lumen of the vessel was in most instances well preserved, no thrombus was found, and only fluid blood filled the vein. No stricture of the vessel was ever found. Schmidt thinks that it is better not to leave the clamps on longer than twenty-four hours, and they should be removed with every possible care and gentleness, so as to prevent any possible laceration of the vessel. He also records seven cases of injury to veins in men in which this method of hæmostasis was applied. The clamps were left on about twenty-one hours, and after they had been removed the cutaneous incision was sutured. No instance of secondary hemorrhage occurred, but one patient died three days after the operation from sepsis.

Niebergall, in considering the various methods employed for closing lateral lesions in the veins, believes that the clamp method is the best. He considers that union of the intima, which is accomplished in twenty-four hours, is quite sufficient to prevent any secondary hemorrhage from occurring after the clamps are taken off. He insists on the advantage we have when using the clamps that no foreign material is left in the wound after the instrument is removed, while in lateral ligature or suture of the vein with silk or catgut the latter has to remain *in situ* until encysted or absorbed.

Simpson long ago closed wounds of the femoral artery in twelve cases by means of acupressure and left the needles in place only forty-eight hours, and it is well known how extensively Péan used this method in all operations, even when large arteries were involved; so Niebergall deducts the conclusion that if large arteries can be closed by clamps in spite of the high blood-pressure and the great elasticity of the walls of the vessel, they can certainly be used to advantage in the case of veins whose loose walls, regular circulation, and low blood-pressure should all tend to render the process of repair easy.

As to the technique of clamping a wounded vein, it is very simple. You simply seize the borders of the opening between the blades of the clamp, and if all issue of blood has stopped the instrument is allowed to remain *in situ* for twenty-four hours under an aseptic dressing. At the end of this time the dressings are removed and the clamp or clamps, as the case may be, are unlocked and gently taken off after separating the blades. The blades are often stuck tightly

together, and if you pull on them you will surely tear the vein, so in this case, after unlocking them it is better to leave them for half an hour or so, and then they may be removed easily, because they will become loosened by their proper elasticity and weight.

When the clamps are off the wound in the integuments is closed with sutures and a tightly applied aseptic dressing is put on.

The presence of clamps is not of any consequence and in no way injurious, and primary union of the skin incision almost always results. But if there should be any reason for fearing that the clamps might injure some of the neighboring parts from pressure, it is easily avoided if you pack sterile gauze well around them.

Niebergall reports fifty-three cases of wounds of the veins closed by the clamp method, eight of which died; and although he attributes the fatal result to other causes, it would seem that at least one patient died from the use of the clamps, and is as follows: While extirpating a lymphosarcoma, the size of a child's head, in the inguinal region, it was necessary to excise the walls of the femoral vein to such an extent that the wound was closed by three clamps. The next day, when the clamps were removed, a hemorrhage occurred. As there was much odor to the wound, the clamps were not allowed to remain, and ligature of the femoral artery was attempted, but the hemorrhage continued and death ensued on the fourth day from a rapidly extending gangrene of the leg.

Now, if in this case no secondary hemorrhage had taken place, and here it is surely the method of haemostasis that was at fault, ligature of the femoral artery would not have been necessary, and probably no gangrene would have appeared.

Niebergall also says that lateral lesions of the veins are best treated by the clamp method, and that by this method the lumen of the vessel is best preserved. Now, such a statement is absurd, because no matter how small an amount of tissue is included in the grasp of the clamp,—and remember that in order to obtain union we are obliged to take up a certain amount,—the caliber of the vessel must naturally be somewhat diminished.

There is always a certain amount of uncertainty in this method, and too much reliance is placed upon the probable closing of the vessel in twenty-four hours. I should strongly advise you never to take any risks with the lives of your patients, and this method is a risky one. We can never know just how long the clamp should re-

main in any particular case, and even Niebergall was obliged to keep them on in several cases for two, five, or even fourteen days.

The presence of the clamps is apt to be a source of irritation to the tissues with which they come in contact, and the wound cannot be closed so long as they remain *in situ*, which consequently is a disadvantage to union of the wound *per primam*, and being left open will expose it to the danger of infection.

There are one or two instances in which clamps à démeur are of value; the first is in vaginal hysterectomy, and the second is the pedicle after nephrectomy, when the former is so deeply seated that a ligature cannot be securely applied. But as these cases are not directly connected with the subject of this lecture, I will not go into further details.

The ideal method of hæmostasis in lateral wounds of the veins is most certainly suture. It satisfies all the requirements demanded by modern surgery,—namely, it restores the lumen of the vessel, it is hæmostatic, it prevents secondary hemorrhage, and it is easy of application.

Broca records the first case of suture of a blood-vessel in his treatise on "Aneurisms." It was done by a French surgeon at the end of the last century, who closed a longitudinal opening in the brachial artery successfully. Gensoul, in 1833, reported two cases of suture of the internal jugular in horses, but inflammation and obliteration of the vessel resulted.

In 1881 Czerny first deliberately performed suture of a vein in the following instance. He performed external oesophagotomy for a foreign body, and eight days later he sutured the common jugular, which had become eroded from a suppurative process arising in the wound. Two days after bleeding again took place, the vein was again stitched, a lateral ligature with pressure was applied, but the patient died in a few days from pyæmia. This case, of course, is not a fair example to go by, as the suture of the vein was attempted under most unfavorable circumstances.

Schede successfully sutured an opening in the femoral vein in the case of a patient who had enlarged glands in both inguinal regions following carcinoma. On the right side the femoral vein was incised while extirpating the mass; a tourniquet was applied and the opening in the vessel was closed with fine catgut sutures, and the sheath of the vessel was also brought together with sutures. There followed

no disturbances in the circulation except the œdema that naturally follows such operations. Other successful cases have been reported by Mayr and Heinicke of the femoral vein, and of the axillary and popliteal veins by Heinlein.

In 1883 Gluck reported his experimental work on dogs and rabbits, but did not conclude favorably of vein suturing, as haemostasis could not be obtained in his experiments with this method.

On the other hand, Horoch came to contrary conclusions deducted from his experiments with vein suturing in 1888. He made longitudinal and perpendicular wounds in various veins and arteries in dogs, and in each instance the vessel was successfully closed with sutures. Three experiments were made on the veins and four on the arteries, and in one case he made a complete section of the femoral artery. In the latter instance six interrupted silk sutures were used, while in the partially severed vessels five silk sutures were used in one, the other being closed by four catguts. In a longitudinal opening of an artery measuring one and a half centimetres, the wound was closed by six silk stitches. A trifling amount of blood came away at each stitch-hole in the vessel walls, which was of more consequence when catgut was employed, but which soon stopped. Pulsation could soon be felt in every case which demonstrated that the lumen of the vessels had remained patent. One animal died three days after the operation, and the vessel was found completely severed and a thrombosis had taken place in both ends.

In the other animals the veins were exsected after a few weeks and an organized thrombus was found in the lumen. Horoch concludes that the ultimate result of suture of the arteries is quite equal to ligature, and is preferable to the latter in large vessels, as obstruction of the vessel from thrombosis is slower and danger from gangrene is less, as collateral circulation has time to take place and be effectual.

In veins Horoch obtained still better results. He operated on the femoral vein twice and once on the internal jugular. The latter he opened perpendicularly and united the ends by five silk stitches; the femoral vein was cut to about one-half its circumference, and this was closed by sutures; then the other half was incised and also sutured, eleven sutures being necessary to close the entire vessel. In another femoral vein a longitudinal opening was made which was closed by seven silk sutures.

One animal died four weeks after the operation from some unknown cause. Examination of the vein showed that the walls were thickened and its lumen so narrowed that only a probe could be made to enter. In the remaining animals the vein was excised at the end of four weeks; the walls were thickened, but their lumen admitted the passage of a good-sized sound. The sutures were partially absorbed, their remains being present both in and outside of the cicatrix.

The better results obtained in suture of the veins than of arteries is believed by Horoch to be due to the greater expansive power of their walls and a lesser blood-pressure within the vessel.

This authority used small, round, straight needles in his experiments, and he also points out that the approximation of the wound edges must be very exact and secure.

Jasinowsky carried out twenty-six experiments with longitudinal and transverse incisions of arteries, and concludes that if sutured they will unite without any obliteration of the lumen and that thrombosis will only take place if a disturbance of the circulation arises. As an important condition for success Jasinowsky points out that only partial injuries which do not include more than one-half the circumference of the vessel are fit for suture; that the edges of the wound must be even and the vessel not too deeply seated.

Now, if during an operation a vein is accidentally injured, the first thing to do is to close up the opening by tamponing, in order to prevent the escape of blood and the entrance of air into the vessel. If the large vein cut runs over the surface of a neoplasm, the opening may be immediately closed if the tumor is pulled upward, because pressure is thus brought to bear upon it and at the same time the vessel is exposed to better advantage.

If the subclavian vein should be injured during resection of the clavicle, for example, Heinicke advises the temporary ligature of the vena anonyma, and then, after compression or temporary ligaturing of the internal jugular and subclavian, the opening in the injured vessel is closed with sutures and the ligatures are removed.

Small, round, straight or curved needles should be used for suturing, because the silk or catgut will obliterate the needle-hole. A needle with sharp edges will cause too much damage, and after the sutures are in place oozing will take place through the stitch-holes.

Horoch believes that silk is the best material to use, while Schede

prefers catgut because it swells when moistened and consequently fills up the stitch-holes. Small catgut becomes absorbed rapidly, and for this reason should be preferred to silk, as no foreign substance will remain in the walls of the vessel.

In suturing it is better to do it accurately, bringing intima in apposition with intima, and adventitia with adventitia; the repair of the wound takes place by a proliferation of the endothelium. Repair will nevertheless occur even if the various coats of the vein are not directly brought in contact with one another, so long as asepsis is carried out perfectly. Thrombosis is not likely to occur if care is taken, and even if the catgut should become detached so as to project into the lumen of the vessel, Petit has shown that this will be absorbed and will not give rise to the formation of thrombus. If haemostasis is complete after the vein is sutured, it may be safely left. We naturally get a few drops of blood from the stitch-holes, but this is of no consequence.

The various kinds of sutures have been experimented on by Mayr on the cadaver. Lateral incisions were made in the veins and were closed with interrupted sutures, double continued suture, the running suture, and quilled suture. Water was then introduced under a high pressure, and its effects on the various forms of suture were as follows: The interrupted suture was useless, as the fluid made its exit between the stitches, while the double continued suture only allowed the water to pass out of the opening when the pressure was very high. The quilled and running sutures were the most successful.

Practically we will only consider the running suture, as the quilled and double continued sutures are very difficult to make, and, besides, they will reduce the lumen of the vessel considerably in size. Therefore I would advise you to only employ the running suture, as its results are good and it is easy to do. The running suture has also the advantage that it does not come in contact with the blood to any extent, which lessens the possible formation of thrombus.

It is a good plan to suture the sheath of the vein over it after the latter has been stitched, as well as the surrounding tissue, because you will give more support to the vessel by this means. If a vein in a limb has been sutured, it is well to place the member in an elevated position, so as to help the venous circulation and prevent stagnation of blood at the site of the suture, and thus avoid the possibility of thrombus.

The parts must, of course, be kept quiet, as a sudden movement might make the suture give way. Secondary hemorrhages, which sometimes occur when the vessel has been subjected to a protracted process of repair, cannot be attributed to the method, as they take place with lateral or complete ligature as well when placed in the same conditions.

From the clinical cases reported by Petit, Stapanjew, Villar, Bracket, and others, we may reasonably assume that the lumen of the vein remains patent after suturing the vessel, and the amount of narrowing that does take place in no way interferes with a good circulation.

In closing, I would formulate a few rules on suturing wounds of the large veins: 1. Suture every longitudinal opening in a vein of anatomical importance, no matter how long the lesion may be, provided that there is no loss of tissue from the walls of the vessel. 2. Use a straight, round needle and fine catgut. 3. A running suture is to be preferred. 4. If possible, bring the three coats of the vessel into good apposition, but this is not absolutely necessary. 5. Suture the sheath of the vein over it, and over this bring the surrounding tissues together by a few interrupted stitches, in order to give the vein as much support as possible.

CASES OF ACUTE, TRAUMATIC ABDOMINAL HEMORRHAGE.

BY JOHN C. MUNRO, M.D.,

Assistant Visiting Surgeon, Boston City Hospital; Instructor in Surgery, Harvard Medical School.

THE following cases are reported because they illustrate a group of symptoms that is apparently associated with severe intra-abdominal hemorrhage, a group that has induced the writer to operate in several of the cases, and that is not usually associated with severe bleeding in the mind of the general practitioner. Of course, with the symptoms there may be the history of an injury that "knocks out" the patient for a few moments, though this is not necessarily the case. But, if together with the history there is associated a slow pulse not always of poor quality, and slight spasm of the abdomen on pressure one has good reason for urging exploration without delay. Where, as in one of the cases, the chest is crushed or there is external bleeding, other factors enter to change the pulse-rate. Furthermore, as soon as these abdomens, filled to distention with blood, are opened the pulse rises alarmingly in the same way that it does if the operation is delayed for twelve hours or more.

The sense of touch will probably locate a ruptured organ more quickly and surely than the sense of sight; this applies as well to cases of ruptured tubal pregnancy.

CASE I.—Fracture of pelvis; rupture of bladder; properitoneal hemorrhage. Peter S., a Danish rigger, thirty-nine years old, while lying on a deck at work, was struck by a spar which broke his pelvis. He was unconscious for a few minutes, nauseated, and had much pain in the abdomen. At entrance to Dr. George W. Gay's service at the Boston City Hospital shortly afterwards he was in considerable shock, but with a pulse of good strength and not rapid. Catheterized and urine found to be bloody. Abdomen tender all

over and flat from umbilicus to pubes. An hour or two later the catheter drew blood and a sufficient quantity of urine.

Operation at once under ether. On reaching the peritoneal space there was a sharp gush of fluid and clotted blood, the peritoneum being pushed back against the posterior wall and stripped from the parietes as far up as the umbilicus. The active bleeding coming from the lower end of the rectus and from around the neck of the bladder was controlled by a ligature around the muscle and packing in the space of Retzius. The peritoneum was then opened, but as there was no hemorrhage or evidence of urinary leakage into the peritoneal cavity, the peritoneum was stitched to the under surface of the rectus and the wound closed except at the lower end, from which the wicks protruded. Examination by rectum showed mobility and crepitus of the pubic bone, the crack apparently passing near the symphysis. On the following day a catheter was tied in. On the second day, the catheter having become plugged, urine was detected in the dressings. A few days later there was considerable pus in the urine and a little blood. By the end of two weeks, as the bladder could not be kept clean, and as the quantity of urine escaping over the pubes was increasing, the bladder was drained through the perineum. In four days the urine ceased escaping into the wound, only to reappear on accidental escape of the perineal drain; eventually, however, about five weeks after the last operation, no urine came over the pubes and the wound closed. Two weeks later the patient was around on crutches, and soon left the hospital in good condition, since which time he has been working steadily.

CASE II.—Penetrating wound of the abdomen; wound of liver. Michael B., a powerful mill hand, twenty-eight years old, while shifting a belt with a broom-handle, which was caught and broken by the machinery, was struck in the belly by the sharp end. The stick entered at the epigastrium, and was withdrawn by the patient, who was brought to the Carney Hospital, where I saw him an hour or two later. He had a strong pulse of 72 and a normal temperature. From the wound a small bit of omentum protruded; the abdomen was distended, but the patient complained of very little pain and gave reluctant consent to operation.

Operation, ether. The original wound was slightly enlarged and the strangulated omentum ligated and removed. This seemed to be all the injury until the omentum was pushed back, when a gush of

blood showed the cavity to be full of blood, fluid and in clots. The opening was then enlarged and the intestines near by hastily examined without showing evidence of any lesion; the incision was carried to the right, and the hand in the belly detected a tear at the extreme edge of the liver on the right. By still further enlargement of the wound the tear in the liver could be grasped by the assistant and the bleeding controlled until the abdomen could be cleared of the clots and blood. Four deep sutures of heavy silk were then passed through the liver, entirely controlling the hemorrhage. After irrigation with salt solution, the wounds were closed with a drain at the extreme right.

As soon as the abdomen was opened the pulse rose to 140 and stimulation was used freely. The patient passed a comfortable night, and on the following day the pulse had fallen to 95, but twenty-four hours later, although there had been no vomiting and the bowels had moved, he grew restless, and at the end of the third day after operation died probably of peritonitis. No autopsy was allowed.

CASE III.—Stab-wound of the liver. George P. F., a large, fat man, fifty-two years old, was assaulted and stabbed in the axilla and abdomen. At entrance to Dr. Gay's service at the City Hospital he was blanched, with a poor, rapid pulse, sweating, in pain, and with short, somewhat increased respirations. There was free bleeding from the axilla and from a horizontal wound a little to the left of the gall-bladder about an inch and a half long, from which blood escaped by the side of the protruding omentum.

Operation, ether. Vertical incision three or four inches long from the right side of the stab-wound through a wall containing fat two inches thick. Throughout the operation the patient grunted with each expiration, so that it was difficult to control the intestines. As much blood, fresh and in clots, came from the region of the liver, the stab-wound was enlarged rightwards, and a wound an inch and a half long and about half an inch from the free border and from the great fissure was seen penetrating the lobe and bleeding freely. The gall-bladder appeared normal in size and tension, but a portion of its upper surface was dark from hemorrhage under the serous covering. A double, heavy silk suture through the liver controlled the bleeding. As the patient was losing ground, the wound was closed as rapidly as possible with a gauze drain packed down to the sutured liver. Stimulation was freely used, and the patient rallied some-

what. Eight hours after operation he was in fair condition and apparently reacting, when he began to bleed through the dressings, the pulse grew rapid and thready, and he died twenty-four hours after operation.

He did not rally after the intermediary hemorrhage enough to warrant interference. Autopsy showed a wound of the gall-bladder as well as of the liver. The axillary wound was deep, but it did not injure the large vessels.

Had the patient been thin and taken the ether well he might have been saved. The increased difficulty of working through a flabby, fat abdomen, and the constant enforced handling of the intestines, increased the shock very much.

CASE IV.—Multiple injuries; abdominal hemorrhage. Joseph F. L., forty-four years old, was knocked from a car and then crushed between the car and a post. Unconscious for a while. At entrance to Dr. H. W. Cushing's service at the City Hospital he was cyanotic, with rapid, shallow respiration; a weak, rapid pulse; diminished breathing, and crackling râles over the left chest; a distended, tender abdomen, with spasm on deep pressure. Nausea, no flatus by rectum, no blood in the urine. During the night he was stimulated, as he was too much in collapse at admission to warrant interference. As he rallied somewhat and the chest symptoms, which were the important ones, were no worse, operation was advised.

Operation, chloroform, atropia to dry the secretions. As spasm persisted under full anaesthesia, the abdomen was opened in the median line and the cavity found full of blood. Examination of the intestines, liver, kidneys, etc., failed to locate the origin of the bleeding, but as most of it appeared to come from the right flank, an opening was made there, and later on in the left flank also. Constant and profuse irrigation with hot salt solution washed out a large quantity of dark blood, but without evidence of any fresh hemorrhage. Both flanks and the pelvis were packed with gauze wicks and drainage tubes. Salt solution and stimulants were used freely, and the patient stood the operation fairly well considering the condition of his chest. On the following day he passed a little gas, vomited only once, and showed no signs of further bleeding. He continued to fail, however, and died about forty-eight hours after injury. No autopsy was allowed.

The case was a desperate one, and operation was advised as a last

resort. There was undoubted injury to one lung and abdominal hemorrhage, but with the other lung in good condition there was a possibility that he might survive if the bleeding could be checked, and, as it proved, he was not apparently much worse for the operation. There was no clue to the source of the bleeding; it may possibly have come from the chest or diaphragm, although it could not be traced to that part of the cavity.

CASE V.—Penetrating bullet wound of the abdomen. James H. C. was shot at close range by a policeman three hours before operation. He was thirsty, pale, and with cold extremities, but he had a regular pulse of about 60, although of poor volume. Just below the ensiform was a round, blackened wound of entrance, and posteriorly the hole of exit through the outer edge of the erector spinae between the eleventh and twelfth ribs. Soon after entrance to Dr. Cushing's service at the City Hospital he vomited, but no blood was found in the vomitus nor in the urine drawn by catheter.

Operation, ether. Incision from wound to umbilicus. Abdominal cavity full of fluid and clotted blood. A small blister was found on the liver, but all the bleeding evidently came from the right kidney or thereabouts. The abdomen was flushed out, the lower portion of the wound closed, and through the upper portion wicks were packed down to the renal region. The posterior wound was then enlarged and explored and the sinus packed with gauze which controlled the hemorrhage.

On the tenth day a mass of omentum as large as the fist was found protruding from the upper angle of the wound at the place of entrance of the bullet; this was carefully dressed, and in two weeks had shrunk nearly to the level of the skin, and three days later it had disappeared altogether, leaving a flat granulating surface that rapidly closed. At the end of ten days from the injury he was discharged healed, wearing a swathe.

CASE VI.—Rupture of kidney; injury to chest. Michael Z., an Italian laborer, fell fifteen feet, striking the left side of his chest and abdomen upon a large stone. He got up and worked a short time, until the pain in the left side compelled him to stop, and he was brought at once to the City Hospital to Dr. Gay's service. At entrance the abdomen was not distended, but tympanitic, except over the bladder and in the left flank, where it was flat. The pulse and respiration were good, and he showed no signs of serious injury ex-

cept that of fresh blood in the urine. The next morning, twelve hours after injury, he was markedly worse. There was a moderately distended abdomen, with tenderness and spasm; increasing pulse and respiration; no flatus; blood still in the urine.

Operation, ether. Incision in front over left kidney. Considerable fluid blood escaped from the peritoneal cavity, and the descending colon was found pushed forward with the posterior peritoneum by an extravasation which extended down into the pelvis and across to the median line. To the outside of the colon was a small rent in the peritoneum, through which the blood oozed from the renal region. This opening was enlarged and several lacerations were found in the lower end of the kidney; elsewhere it felt normal. No odor of urine was detected. The perirenal space about the lacerations was packed with gauze, controlling the bleeding, the peritoneal cavity washed fairly clean with salt solution, the lower two-thirds of the wound closed, leaving the upper portion packed with gauze, making a well from the kidney to the surface. The patient was considerably shaken up by the operation, but responded well to stimulants.

He did well for two days, when a pneumonia developed in both lungs; the râles could be felt as well as heard on both sides; he passed urine involuntarily, but none came through the wound. Atropine was pushed to its limit and stimulation continued. He then improved for three days, when the pneumonia increased and atropine was again pushed. About this time he also developed a marked pneumo-pericarditis in addition to his other troubles. Eight days after operation the wicks were all out and no urine had escaped from the wound. About a week later, however, the urine contained pus and casts, and it began to escape from the wound; he continued to gain nevertheless, up to three weeks after the operation, when a sharp rise of temperature heralded a fresh attack of pneumonia in the left lower lobe. By dint of splendid nursing he pulled through these various attacks and was left to contend with only a urinary fistula at the site of the original wound. Under chloroform, eight weeks after entrance, a lumbar opening was made to drain the leaking kidney, when the anterior wound rapidly closed, followed in a few days by the posterior. Eleven weeks after injury he was discharged strong and in good condition.

CASE VII.—Rupture of spleen and kidney. Frank S. E., forty years old, was struck by a falling tree and brought at once to Dr.

Gay's service at the City Hospital. The symptoms all pointed to injury of the chest, where there was pain and tenderness. He had a pulse of 70, of good volume; no distention of the abdomen, but very slight spasm, more marked on the left. Four hours later there was beginning distention, with more spasm and dulness in the flanks. The pulse remained at 70, but of poor strength. The color was bad, and the hands were cold. No blood in the urine.

Operation, ether. Spasm persisted under full anæsthesia. A median incision was made to admit the hand, and the abdomen found full of blood, fluid and in clots; the intestines were contracted to the size of a lead-pencil. To the touch the spleen felt rough, the liver smooth; no fæcal odor was detected. A second incision was then made over the spleen, which was lacerated over a space two inches square; as it was quite movable, it was tied off with silk and removed. In clearing out a large mass of adherent clots in the left flank, a sudden gush of blood followed from the region of the kidney, which was also found ruptured, hanging by its pedicle free in the abdominal cavity. A clamp was then placed on the pedicle, but no attempt was made to remove the organ, as the patient was pulseless. Some more blood and clots were scooped out, the abdomen flushed out, and the median wound closed. The splenic region was packed with gauze and the lateral wound partially closed. About three pints of salt solution were given under the breast, besides strychnine, etc. At the close of the operation, which lasted only twenty minutes, the patient was in profound shock, but rallied as soon as he was put to bed. During the night he continued to improve with a rapid pulse of fair strength; there was no vomiting nor distention, and he took nourishment well both by mouth and by rectum, but about twenty-four hours after operation he began to lose ground, and steadily failed until death, twelve hours later.

Gynaecology and Obstetrics.

APOPLEXY DURING PREGNANCY.¹

CLINICAL LECTURE DELIVERED AT THE CHARITÉ HOSPITAL.

BY PROFESSOR C. GERHARDT,

Director of the Second Medical Clinic and Professor of Special Pathology and
Therapy at the University of Berlin, Germany.

GENTLEMEN,—The patient whom I bring before you this morning is here for the second time, and, although she is but twenty-five years old, came both times because of apoplectic attacks, the apoplexy on each occasion coming on during the course of a pregnancy. The case is an extremely interesting one because of the youth of the patient and the circumstances, and gives us the opportunity to speak of the relation between pregnancy and apoplexy.

Of her personal and family history we have not been able to learn much, because of her aphasic condition. From the history obtained during her former stay in the hospital we gather these details: Her father died suddenly at sixty, whether from apoplexy or not is not clear. Her mother died at forty-six of a pulmonary affection; she has five brothers and sisters, who are all healthy.

She herself worked for a while in a laundry, and afterwards sewed for a living, but claims never to have been in want and never to have drunk to excess. She has had no venereal disease. In November, 1894, about four years ago, when she was twenty-one therefore, she was admitted to the First Medical Clinic suffering from the remains of a left-sided hemiplegia, which came on suddenly during the preceding March, at which time she was in the fifth month of a first pregnancy. One evening she was taken with an attack of vom-

¹ Reported by James J. Walsh, Ph.D., M.D.

iting accompanied by vertigo, with loss of power in the left leg and arm, and was delivered some three months later of an immature child, which died after ten days without any special signs of disease, seemingly from lack of vitality.

She was transferred to the medical clinic for the treatment of her hemiplegia after her confinement, and under the use of electricity her condition improved very much, so that after some months she was able to leave the hospital in very good condition under the circumstances. She could use her left arm well, her foot dragged slightly and the reflexes were exaggerated, but there were no contractures, except some stiffness in the ankle-joint. It is noted that when she first came to the medical clinic at that time, she had a systolic murmur at her apex. When she left the hospital this had completely disappeared and the heart sounds were absolutely clear and normal.

A couple of weeks ago, during the course of a second pregnancy,—in the eighth month this time,—she had a sudden loss of power in her right arm, with aphasia. The loss of power spread later to the right leg. The attack came on absolutely without any premonitory symptoms. We have, then, a remarkable case of a young woman of twenty-six who has had two apoplectic attacks. You may note in passing that her sensorium is undisturbed, and that she is fully in the possession of her senses, and will not, with womanly wilfulness, allow herself to be made older than she is. Note, also, that her aphasia is passing off. This is, I think, the first time that I have heard her speak spontaneously without being questioned. Usually she has to be encouraged to make a special effort.

She had her first apoplectic attack at the age of twenty-one, while, of course, apoplexy is, as a rule,—to which exceptions are rare, though they occur,—a disease of the old. Apoplectic attacks are even rarer in early adult life than in children, as in children there are occasional cases of cerebral hemorrhage noted. In young adults apoplexy occurs oftener in women than in men, and it is due oftener to embolism or thrombosis than to hemorrhage. It is always to be borne in mind that an apoplectic attack in young people usually means syphilis, and careful investigation should be made with this idea in mind.

Besides the three causes mentioned,—hemorrhage, embolism, and thrombosis,—there are others that may give the symptoms of an apo-

plectic attack. Tumors, for instance, may first reveal their presence by a sudden attack of partial or complete unconsciousness, after perhaps vomiting and vertigo, and the patient wakes up to find power lost in one side of his body. Besides this, there may be a congestive apoplexy, the fluxionary apoplexy of the older writers, a loss of consciousness, followed by paresis of the extremities as the result of intense brain hyperæmia, which by an increase of intracranial pressure interferes with cortical nutrition and function. Then there may be a serous apoplexy from over-production or accumulation, for some reason, of cerebro-spinal fluid within the ventricles, and there may be a true apoplectiform attack from nervous causes, great emotion, sudden shock or fright, interrupting nervous conduction for a time, the functions only being restored gradually.

These last-mentioned causes are extremely rare, however, though tumor is not. The best diagnostic means we have for brain tumors is the ophthalmoscope. It should always be used when there is the slightest suspicion of tumor being present. In by far the greater number of brain tumors,—from ninety-two to ninety-three per cent., I believe, are the statistics,—choked disk is present. The increased intracranial pressure leads to a congestion and œdema of the end of the optic nerve, that becomes easily visible to the ophthalmoscope as a swollen prominent disk with indefinite edges. Where choked disk is not present, we can almost exclude tumor, the cases are so rare in which it does not occur. When confirmed by other symptoms the diagnosis becomes extremely easy. Here we have no choked disk, and can, therefore, practically exclude tumor from the case.

In young people, as I have said, and especially in young women, we must think of embolus. Its most frequent source is, of course, the left heart. We find that the patient had a heart murmur on her admission before, and we find a heart murmur now, so that the question of embolism would seem easily settled. But when she left the hospital before her heart murmur had disappeared, so that the interesting question insinuates itself, Was there ever really a heart lesion present?

This question of a valvular lesion in the heart is not as easily settled as students and young physicians often seem to think. Time was when a physician put his ear down to a chest wall, heard a murmur, and diagnosed a lesion of one of the valves of the heart. We have

studied the question of valvular lesions much more since then, and we know that in very many cases we cannot be sure. Certainly, with the disappearance of the heart murmur later in the case and the failure of any other heart symptoms, we would be tempted rather to think that there had been a functional heart murmur, than that a valve which had been roughened or sclerosed enough to give an organic murmur, had later undergone changes in the direction of improvement, which had led to the disappearance of the murmur. Certainly that is not the character of the alteration that valvular lesions usually take. Heart murmurs due to an organic lesion sometimes disappear as a patient grows weaker, but very, very rarely as the patient gains strength.

It is true, when we can demonstrate some enlargement of the heart, and there is an accentuation of the pulmonary second sound and a systolic murmur at the apex, it seems clear that we have a mitral lesion; yet I think I have seen all these symptoms in patients where the course of the case led me to believe afterwards that we had not had to deal with a valvular lesion at all. When there is rheumatism or chorea in the personal history, then other elements of assurance as to the organic character of the murmur are added, but even in such cases we cannot be sure.

In this case slight enlargement of the heart was noted to have been present during her first stay in the hospital, but as that is usual in pregnancy, or rather because of circumstances it appears to be so, it must not be permitted to have much significance for us. We cannot, then, definitely conclude that the apoplectic attack at that time was due to an embolus, but we are free to make our diagnosis according to the conditions that we find at present.

Physical examination of her thorax shows nothing abnormal in her lungs. Her apex-beat is in the sixth intercostal space just outside the nipple line. As she is in the eighth month of her pregnancy, and her breasts are already fuller than normal, the apex-beat is distinctly farther out than it should be. Percussion shows that while heart dulness does not begin as high up as normal, it extends slightly beyond the sternum to the right. The vault of the diaphragm is flattened by the increased pressure within the abdomen, at the same time that the diaphragm, as a whole, is forced upward, so that the heart, instead of hanging suspended by its base more or less vertically, is lying somewhat on its side on the diaphragm.

This gives the broad low heart dulness usually found in pregnancy, and which has by certain French writers been taken to indicate that the heart is enlarged, but which really only means its displacement because of the altered position of the diaphragm. On auscultation we find a short systolic murmur at the apex, the pulmonary sounds are normal, and the aortic and tricuspid give perfectly clear tones.

There is not much in the heart, so we turn to the kidneys. The urine is of a pale-yellow color, of about normal amount, and contains small quantities of albumen. This is at once of interest, for cases of true apoplexy, hemorrhage into the brain from a ruptured artery, are caused much oftener than is thought by contracted kidney. With a urine pale yellow in color, plentiful in amount, and a small amount of albumen, most of the urinary symptoms of contracted kidney are present; its specific gravity is, however, too high. In contracted kidney, and I use the term advisedly to signify the sclerotic atrophy of the kidney that comes at the end of chronic nephritis of all kinds, in order to avoid specification of its origin, the specific gravity of the urine is always low,—1003 to 1008. Here we have 1015, 1025, 1021, 1018, 1024, 1017, 1022, a most variable specific gravity, but not low and yet not too high.

The albumen might be due to the so-called kidney of pregnancy, an affection of the kidney due to the interference with its blood-supply by the pressure of the gravid uterus. There is a fatty infiltration of the kidney epithelium due to malnutrition, with albuminuria and at times dropsy, especially of the legs. Here we have no oedema, and with urine in normal amount and of about normal specific gravity there can be no question of severe nephritis. Albuminuria alone, however, when it occurs during pregnancy, and careful search has revealed no casts in this case, usually does not signify an organic lesion of the kidney, and, as a matter of fact, the kidney of pregnancy, under ordinary circumstances, always returns completely to the normal.

The symptoms of her palsy may lead us to the more exact localization of the lesion, and so give us a clue also to the nature of the pathological process. You see that her head is turned persistently to the right as she lies in bed. I have her sit up, and the same thing is noticeable. I ask her if she always keeps her head turned this way, and she says, yes. Her aphasia prevents her from telling me, in

answer to my question, why it is that she turns her head this way. Usually turning of the head is associated with a corresponding turning of the eyes, deviation conjuguée of the eyes and head, as the French have called it. It has then been thought to have some diagnostic significance, the look being towards the lesion—i.e., to the side on which the lesion is—when it is in the cerebral hemispheres, and being away from the lesion—i.e., towards the palsied members—when it is at the base of the brain. Here we do not need the hint this might give us, because the existence of aphasia shows that the lesion is on the left and has affected the cortex.

When she attempts to talk, or, at my request, tries to laugh, you see that there is palsy of the *facialis*. Her mouth is pulled to the left, and the *nasolabial* fold is deeper than on the right. She closes both eyes well, however, and it is as difficult to lift the lid of one as of the other, and she wrinkles her forehead equally well on both sides. The uppermost branch of her *facialis* is, as is usual in palsies of cerebral origin, not involved.

When I have her put out her tongue, it is deviated to the right. That it is a true deviation you can see when I put my thumb here on the *symphysis menti*, the little fossa here in the chin, for my thumb indicates clearly the median line. At times in such cases the appearances are deceptive. The paralysis of the *facialis* leads to a contortion of the face when the mouth is opened, which pulls the angle of the mouth and with it the fossa here in the centre of the lower lip to one side. When the tongue is projected, then, though the projection may be perfectly straight, the impression may be produced that it goes to one side, because the usual position of the landmarks for the centre of the lips is disturbed. By getting the position of the *symphysis menti* clear no chance for this illusion is left.

The tongue is deviated towards the right, as is usual in paralysis of the right side. In its projection the tongue is not *pulled* forward, but *pushed* forward; the deviation to the right, then, is not due to any superior strength of the muscles on this side, but to the fact that the *genio-hyoglossus* of the left side as it pushes the tongue forward does not meet with its usual resistance from the companion muscle on the right, and so pushes the tongue beyond the median line in the direction of lesser resistance. The subject is important to have clear once for all, because there are a number of cases of so-called crossed paralysis of the *facialis* and the extremities which seem to rest on no

better foundation than a misunderstanding of the result of one-sided palsy of the tongue.

As to her extremities, her right arm is stiff and slow in movement, but has gained very much in power and motion in the last few days. Her right leg is also slow and awkward, but it, too, has improved a good deal over the condition it was in when she entered the hospital. Both her patellar reflexes are exaggerated, but especially the left, and her Achilles phenomena are both lively, though here, too, the left is more marked. The hemiplegia of four years ago has left a certain amount of spasmodic contracture in her left leg, hence the exaggerated tendon reflexes. The lively reflexes here on the right show that the prognosis for complete recovery of the usefulness of her right leg is not favorable. The exaggerated reflexes that occur shortly after an apoplectic attack have very little significance, and are nothing more than a manifestation of the irritative condition that exists in the central nervous system just after the attack. But the heightened reflexes that develop after two or three weeks, and persist, are a sign that the inhibitory influence of the higher cortical centres is interfered with, and that the nervous connections between it and the lumbar centre for the leg reflexes are organically impaired.

As to her aphasia, it is not hard to determine its character. She understands what is said to her, she knows the use of objects, she is able to use certain monosyllabic words correctly and readily, and she can repeat others, provided they are not long ones. She has simple motor aphasia. The lesion, then, has affected the cortex in the region where the speech centres are situated, as well as affecting the motor-tracts somewhere in their course from the cortex to the periphery.

This is the second time that she has had an apoplectic attack during pregnancy, so that we naturally come to the question, Has the pregnancy anything to do with it? French physicians especially have claimed that the alterations in the circulation incident to pregnancy were sufficient of themselves to create a certain tendency to rupture of arteries where they were unsupported by firm tissues and for any reason weak. They say that the mother's heart must work for two beings, and so has a double task. They point to the physical signs of this laboring heart in certain manifestations in the arteries that are well known.

The hypertrophy of the uterus and the dilatation of its arteries give rise to a systolic murmur easily heard over the uterus. This, the

so-called placental bruit, because it was supposed to be due to blood streaming through the placental sinuses, is probably only due to blood friction in the enlarged tortuous arteries of the uterus itself. A similar sound may be heard over the enlarged thyroid in Basedow's disease, where a like condition of the arteries exists. Such a sound may be heard in children's heads before the fontanelles close,—that is, up to the sixteenth or eighteenth month of life. It disappears after the closure of the fontanelles or when there is an increase of intracranial pressure, as in hydrocephalus. It is evidently due to the blood streaming through the carotid in its confined bony canal at the base of the skull, into the diminished pressure of the intracranial space.

There is, besides, a double tone to be heard in the femoral arteries in most cases in the second half of pregnancy. This does not exist in this case, and there is often, also, a pulsation of the spleen and liver which we have not here. Besides this, the veins of the legs particularly often enlarge and become varicose, and oedema may develop even where there is no albuminuria. These symptoms are, however, not manifestations of increased heart action, but of hinderance to the return circulation, and may occur with any tumor.

The other circulatory phenomena have been attributed by some to hypertrophy of the heart, that develops during pregnancy. French writers claim that a certain amount of this hypertrophy persists even after the pregnancy is over, and that especially after repeated pregnancies there is a persistent hypertrophy that is very noticeable. I was very much interested in this subject years ago in Jena and Wurzburg, and made a large number of comparative heart measurements at a time when the mortality of confinements in hospitals was higher, and more favorable opportunities and more abundant material for such observations were at hand. I could not find, however, any secure anatomical basis for the so-called hypertrophy of the heart in pregnancy. If the hearts of those who died towards the end of pregnancy, or during or just after their confinement, were larger than normal, it was certainly by a very minimal amount; so that the supposed clinical demonstration of considerable enlargement was certainly an error.

The opinion is, as we have said before, founded on the altered position of the heart and diaphragm during pregnancy, so that heart dulness is demonstrable to the right of the sternum. A confirmatory

physical sign of the supposed hypertrophy is the tendency to *pulsus celer*, and the double tone, not unlike that noted in aortic insufficiency, that can be found in the femoral artery in such cases. This is due to the altered conditions of intra-abdominal pressure, their effect upon the abdominal aorta, and the consequent increased energy demanded of the heart to send blood through the circulation. There may be a consequent element of cardiac hypertrophy in it, too, but it is but slight and temporary if it exists, certainly not permanent.

Pregnancy does not, then, cause a tendency to genuine apoplexy, that is, to hemorrhage into the brain. There is practically no increased blood-pressure, and consequently no exaggerated intracranial arterial tension. The cases that occur are usually embolic in character, the altered composition of the blood during pregnancy giving, perhaps, a reason for coagulation processes, the left auricula cordis being a favorite seat, portions of the clot finding their way to the brain. Of course, if there already exists a heart-lesion there is no reason to evoke the more complicated process, and a portion of a vegetation from a heart valve forms the embolus.

That the affection is embolic in character its rapid amelioration shows. Where it comes on shortly before confinement the symptoms usually pass away in a very short time after confinement. My last case in private practice recovered after a very short time, and only some slight symptoms of aphasia remained. Here, as you see, improvement has already commenced: she is able to use her hand, though as yet but awkwardly and slowly, and she has recovered movements in her leg. Almost the first words that she has spoken beyond yes and no you heard her say this morning. Curiously enough, they were a correction of her age as we had it recorded; she is twenty-five, not twenty-six. Under circumstances that would be apt to perpetuate an error of this kind, no woman would keep silent if it were at all possible to talk.

This rapid improvement speaks strongly for embolism, the embolus being situated in a branch of the left Sylvian artery, and so interfering with the blood-supply to the internal capsule on this side, as also with that to the insula Reilii and cortical speech centres. The disturbance in the internal capsule gives rise to the hemiplegia, and that on the cortex to the aphasia. When she was attacked by hemiplegia before it was on the other side, and there was no aphasia, because the speech centres are situated on the other side of the brain.

It is curious enough that the other side should be affected now, and there is no good reason to account for the succession.

The fact that another lesion had preceded might, in a case where such marked improvement had not taken place, have given rise to difficulties in diagnosis, for all four extremities would have been palsied, and it would be difficult to say whether the new lesion had affected the extremities affected before or not. Fortunately we are not put in any such dubious circumstances. Palsy of all four extremities might follow a lesion in the pons, but this is much rarer than has been thought. It might occur also from a lesion of the medulla, but lesions of the bulb are apt to be rapidly fatal from involvement of essential vital centres. Such lesions have, however, occurred without proving immediately fatal. Then, in case of hemorrhage into the brain, it is by no means so rare to have hemorrhage occur simultaneously, or, at least, with but a small interval, on both sides of the brain. Such cases are often seen at the autopsy, though one of the lesions may have caused no symptoms, or at least none that could be noticed because overshadowed by others.

As to the therapeutics of such cases, there is very little to be done except put the patient in favorable circumstances for recovery of function by absolute rest, judicious nourishment, and symptomatic treatment, if any seems indicated. After improvement has set in in this way, gentle electrical treatment may be instituted and massage employed; these measures will help considerably to the more rapid restoration of muscular function in the palsied limbs. As a good therapeutic rule, however, where lesions of the central nervous system of any kind occur in young patients,—*i.e.*, before middle life,—we must think of syphilis, and must try the effect of syphilitic treatment. We have not much reason to suspect syphilis here. Infection is denied, and there is nothing in the history except, perhaps, the premature birth of a child that did not live at her first confinement. This might well have happened from other reasons, especially in view of her palsied condition during her first pregnancy. We shall continue for a time, however, the use of the iodides, in order to derive any benefit that may be possible from their specific or resolvent properties.

OVARIOTOMY.

CLINICAL LECTURE DELIVERED AT THE JEFFERSON HOSPITAL.

BY E. E. MONTGOMERY, M.D.,

Professor of Gynæcology in the Jefferson Medical College; Gynæcologist to Jefferson and St. Joseph's Hospitals; Ex-President of the Philadelphia Obstetrical and of the Pennsylvania State Medical Society, etc., Philadelphia, Pa.

GENTLEMEN,—The patient I bring before you to-day is forty-nine years of age, a colored washwoman, whose parents died at an advanced age. She had several brothers and sisters die in childhood, and presents no history of any hereditary or family sickness. She has had most of the diseases of childhood. Puberty occurred at fourteen; menstruation regular and painless. Married at nineteen, she has given birth to six children, and has had one miscarriage. The youngest child is fourteen years of age. Thirteen years ago she had pleurisy on the left side, and indirectly following this had some obscure pelvic trouble, an abscess and discharge of a large quantity of fluid through the bowel. She was confined to bed at this time for three months, and did not fully recover for seven months. The menopause occurred seven years since. Five years ago she suffered pain in the left side, which directed her attention to an abdominal growth. The pain soon ceased, but the tumor gradually increased in size until the present. The increase in size has been more marked during the past year. Her heart and lungs are normal; her urine is acid, has a specific gravity of 1026, and contains neither albumen nor sugar. She has a large distended abdomen; this distention is symmetrical. No œdema of the lower extremities or other portions of the body. The absence of œdema enables us to eliminate the possibility of its having arisen from either renal or cardiac disease. There is, however, a fluid accumulation in this abdomen. Is it a cyst or is it an ascites? That it is not ascites, or free fluid, is evident from the fact that we are unable to find any resonance until we get well above the line of the tumor. The resonance is absent over the most prominent

portion of the growth. Of course, we must remember that there is an element of doubt in these cases. The patient may have a large accumulation of fluid, with shortening of the mesentery, to such a degree that the intestines cannot float to the surface. We may have a thickened peritoneum resulting from the long continuation of the fluid. Such a possibility must be kept in mind. From the general outline of this distention, we are pretty certain we have a cyst rather than free fluid. You see the distention stands up prominently below; the abdomen is not flattened out as we would expect to find in mere fluid in the peritoneal cavity, so that we have no hesitancy in saying that we have here to deal with a cyst. The operation we propose to do on this patient was introduced to the profession in 1809 by Dr. McDowell in the backwoods of Kentucky. The legend is that when he performed this operation there was a mob outside ready to do him violence for what they considered an unjustifiable procedure. A friend of his succeeded in persuading them to wait until the operation was done, when, if the patient would live, the doctor was to be spared, and if she died, they could wreak their vengeance upon him. You can appreciate the courage required to do an original operation under such circumstances. This was about as much sympathy as was afforded to the earlier operators in this field. Among the earliest operations done in this city were those of W. L. Atlee, who was called by the professors in the medical school at that time a "belly-ripper," and was denounced for doing unjustifiable operations. Our operation consists in making an incision through the skin and fascia down to the peritoneum. In this case adhesions occur between the cyst and peritoneal surface, so that I have opened directly into the cyst. We now dissect out the sac, tearing it off from the peritoneum and doing this under the eye. In dissecting out this sac, we examine carefully over its anterior surface, that the bladder is not adherent and becomes injured. Having removed the sac and ligated its pedicle, I wash out the cavity with a normal salt solution. We examine then, to see if there is any trouble in the ovary. As I find no enlargement of it, and the woman acknowledges to be forty-nine years of age, we will not disturb it. She evidently is much older, as menstruation has ceased a number of years, so she is not likely to have trouble in the ovary if it has not yet shown itself. We examine the peritoneal cavity for any bleeding points, as we have had extensive adhesions. We want to make sure that all hemorrhage is arrested

before the abdominal wall is closed. I introduce a suture in such a way as to gather up the raw surfaces from which there is some bleeding. As the patient is very much prostrated, we raise up the foot of the table in order to throw the blood into her brain. As we look into this abdomen we see the vessels very greatly distended, showing that the removal of the pressure has interfered with the return circulation. We fill up the abdominal cavity with normal salt solution, which serves practically here as a transfusion. Sutures are introduced. This is the second ovariotomy that has been done in this hospital to-day. A section of the class had an opportunity to see an ovariotomy this morning, in which the cyst was smaller, and was situated in the pelvis. Part of the cyst contained pure fluid, while part of it was purulent. Having introduced our sutures, we will remove the gauze and again carefully examine to see if there is any bleeding. The parts are perfectly dry, notwithstanding there have been extensive adhesions. We will close the wound without drainage. We will let the patient down gradually, otherwise she might suffer from syncope. This patient shows you the difficulty sometimes experienced, even during an operation, in arriving at a diagnosis. While I felt perfectly certain this was an ovarian cyst, yet when we opened into the cyst directly, without being able to differentiate its walls, it was with a sensation that we might possibly find we were mistaken. I passed my hand into the cavity at once, and as we did not come in contact with the intestines, I recognized our diagnosis was correct and the adhesions from the cyst had obliterated the line of demarcation between it and the peritoneum. The method we have pursued here is the recognized method of dealing with cases of adhesions to the anterior wall. The adhesions are dense; we are unable to differentiate and determine just exactly when we reach the peritoneum. The better plan is to open the cyst, draw out its central portion to invert it, and, if the adhesions are not too dense, you are able to pull the cyst off from the walls as we did here. While this patient has had a very large tumor, the intra-abdominal pressure is removed, and in order to compensate for it I propose to put some pads beneath the bandage, so the vessels will be supported by pressure upon them. This patient will be put to bed and her limbs bandaged in order to cut off the unnecessary distribution of blood and confine it to the more immediate sections, recognizing that the heart's action is feeble, and that it will be unable to drive the blood through the

vessels and bring it back again; so we compensate for this by bandaging the arms and legs, thus supporting the circulation in the extremities and increasing the amount of blood that will be sent to the heart, lungs, and brain. The operation we have done is usually known as ovariotomy. This term is rather a misnomer, as it does not convey the idea of what is done. The term otomy, you know, means to cut into, and while we have in this case removed the tumor, ectomy would be the proper termination, so ovariectomy or cystectomy would be preferable to ovariotomy. This term has been so long used, however, in connection with the operation, that it does not now seem desirable to change its application. The first step in the operation, as you have seen, was to cut through the abdominal wall. Ordinarily, in making this incision, we are careful to recognize that we have cut through the aponeurosis of the muscle, transversalis fascia, and peritoneum. Any bleeding vessels are secured before the peritoneum is opened. In this patient the abdomen having been long distended, the abdominal walls have become thinned, the muscle wall is atrophied, and the peritoneum so closely adherent to the cyst wall that it was not recognized until the cyst was opened. The adhering peritoneum shut off the possibility of this fluid entering the peritoneal cavity, so the cyst could be emptied without any difficulty. In making an incision up to the umbilicus I did it partly with the idea in view that, as the tissues there are all adherent together, I would be the better able to separate the peritoneum from the cyst wall. As we drew the cyst out, we were at once enabled to pull off the peritoneum and to separate it from the surface of the cyst. The second step, after opening the peritoneal cavity, is to empty and remove the cyst. This step takes into consideration the treatment of adhesions. Ordinarily we are able to introduce the finger or hand into the abdomen, pass it around the cyst, and thus determine the mass of adhesions if any are present. If not, the cyst is emptied at once by trocar. The fluid is evacuated, and we draw out the cyst until it is completely emptied and withdrawn. Adhesions are seen and separated as the cyst is drawn out as we proceed, by simply sponging the parts off from the surface of the cyst, pushing up the intestines and omentum. Where the adhesions are firm and of long duration, it may be necessary to use scissors or knife. In such cases it is important to watch carefully that we do not injure the intestines, and occasionally we find adhesions so close that we cannot separate the intestines from the

cyst wall, or to attempt to do so would injure the intestine and necessitate a removal of a portion of it. Rather than do this, it is preferable to cut away the cyst, leaving a portion of it in contact with the intestines, taking the precaution, however, to remove the lining membrane of the cyst so that no portion of the secreting surface shall be left in the peritoneal cavity. If a number of loops are adherent in this way, we may cut through the portion of cyst remaining between the loops, separating them, leaving a patch upon each loop of intestine. This produces no subsequent unpleasant effect. Probably the patient will recover as well as if no adhesions existed. Having separated the adhesions and withdrawn the cyst, we have now the consideration of the treatment of the pedicle. This is that portion by which the cyst is connected with the body of the patient and through which the vessels pass for its nourishment. This pedicle may sometimes be quite long, consisting of a band of peritoneum, of broad ligament, or a Fallopian tube that is drawn out. Through this we have passing branches of the ovarian artery, supplying the cyst wall, and these vessels in large cysts are at times quite large, necessitating care in ligation to prevent the possibility of subsequent hemorrhage. In ligation of the pedicle, we will be governed somewhat by its length and thickness. We ordinarily pass a ligature with silk, catgut, or other material by a carrier through the centre of the pedicle, twisting the ligature and tying it in two portions. Where the pedicle is broad and cannot be readily tied in two portions without too much traction upon its sides, it may be tied in several sections. It is well to have these loops of ligature interlock, so that there will be no possibility of the pedicle splitting and hemorrhage resulting from below. We need not wait until the pedicle has been secured before we remove the cyst. We may grasp the cyst by the pedicle forceps and cut away above them. If large and heavy, this does away with traction upon the pedicle. The cavity can then be cleansed, and we proceed at our leisure to ligation of the pedicle. Having ligated the pedicle and removed the cyst, we relax it so we can see if there will be any bleeding, as sometimes vessels will not bleed when the tissues are put upon the stretch that will lead to hemorrhage as soon as relaxation occurs. Having secured the pedicle, we then proceed to the consideration of the other ovary. This should be a method of routine. Never close the abdomen without examining the other ovary, as it may be the seat of a cyst of considerable size, which would otherwise be over-

looked and necessitate a subsequent operation. If the ovary is in a healthy condition it should be undisturbed. If a number of small cysts are found in it, these may be punctured or the diseased portion of ovary may be resected, leaving sufficient amount of ovary to perform its functions. The next step is the toilet of the peritoneum,—washing out the cavity if necessary with normal salt solution and drying with sponges; examination for adhesions and hemorrhage, for tearing of the parietal peritoneum, for injuries of intestines, and so on. If there is much bleeding the peritoneal cavity may be irrigated with hot normal salt solution. This has a good effect as a stimulant to the patient; coming in contact, as it does, with the large peritoneal surface, it cleanses the cavity, and by the heat decreases the tendency to bleeding. If the patient has lost considerable blood and is very much shocked, it would be good treatment to fill up the abdominal cavity with a normal salt solution; in this way supplying the vessels, we practically perform a transfusion. After sponging out the fluid, examine the parts and secure any bleeding vessels, suture over points where extensive adhesions have been present, and thus decrease the tendency to bleeding and favor a better condition following the operation. It is preferable in using suture to use an aseptic catgut. In every operation we have to consider the advisability or necessity for drainage. Shall we or shall we not drain? We should not drain in any case in which there is nothing to be removed. You should not drain where the peritoneal cavity is likely to remain dry. Drainage should be used wherever there is considerable oozing, or in cases in which there has been extensive denudation of the peritoneal surfaces with probability of infection from purulent discharges. We may either drain by the glass drainage-tube or by what I prefer in the hospital, the use of the gauze pack. For sutures we may use silk, catgut, or silkworm-gut, according to the indication of the case or preference of the operator. In this patient we will use a single row of sutures, making sure that we bring together the skin, aponeurosis, and peritoneum. After we close the wound we cleanse the surface and apply a dressing; the latter consists of several layers of sterilized gauze and cotton, held in place by strips of plaster and pieces of tape, and over this a bandage. She will be placed in bed and surrounded by hot bottles, the limbs bandaged to increase the amount of blood thrown into the trunk. The foot of the bed is elevated to make it more easy for the heart to send the blood into the brain, and stimu-

lants will be given as indicated. The most valuable stimulants in such cases are strychnine, digitalis, transfusion of normal salt solution, the use of brandy by the rectum, and, where the patient is much shocked and needs a stimulant to the nerve-centres, the use of ice suppositories. After she recovers from the shock of such an operation, without much trouble subsequently, convalescence should be rapid. It is important, of course, to watch carefully the first twenty-four hours with regard to the effects of the ether, as the kidneys may be defective in such a case, even though examination of the urine has failed to disclose it. We should also watch for the possibility of secondary hemorrhage. While we have secured the pedicle carefully, yet there is always a possibility that a ligature may slip or the outer portion of the pedicle become relaxed, and permit a vessel to slip back. The next matter of interest is the condition of the digestive tract. We give this patient nothing by the stomach for the first twenty-four hours, endeavoring to permit her intestinal tract to recover itself before nutrition is administered. We then give her beef-juice, liquid peptonoids, a cup of tea or coffee, increasing the amount of nutrition as the stomach is able to take care of it.

Ophthalmology.

THE SIMULATION OF BLINDNESS AND ITS DETECTION.

CLINICAL LECTURE DELIVERED AT THE CHICAGO POLICLINIC.

BY F. C. HOTZ, M.D.,

Professor of Ophthalmology in the Chicago Polyclinic and of Ophthalmology and
Otology in Rush Medical College, Chicago, Illinois.

GENTLEMEN,—The patient whose eyes you have examined in the dark room came to me a few days ago with the following story: Three months ago, while he was working in the basement of a new building, a small piece of wood fell from the third story and struck him over the left eye. The blow did not knock him senseless, nor did he afterwards show any signs of head trouble; the eyelids were swollen and painful for about one week, and when he could open the eye again he found the sight almost gone. As the sight has not come back, he wants to obtain the opinion of experts as to the nature of the injury, because he intends instituting a law-suit for damages against his employers.

My examination the other day convinced me that this was a very interesting and instructive case of simulated blindness; and to induce the patient to come here I told him I wished to make another examination before I gave my opinion.

You have seen the patient could barely count my fingers at one foot distance; but upon examination of the eye you have found the refractive media clear and the eye-ground of a perfectly normal appearance. I called your attention particularly to the fact that the optic nerve looked perfectly healthy and in every respect like that of the right eye. This is a very important fact, because it furnishes

positive proof that the optic nerve was not injured by the blow. You know violent blows upon the head may cause a fracture of the osseous ring of the optic foramen and a partial or complete rupture of the optic nerve at that place, with partial or complete loss of sight. If you examine such an eye in the first weeks after the accident, you find the ophthalmoscopic appearance of the optic disk still perfectly normal; but if you examine it again after two or three months, the ophthalmoscopic picture of the papilla will present unmistakable signs of atrophy of the optic nerve. In the present case the accident occurred three months ago, and we find no signs of optic-nerve atrophy; it is therefore not probable that the blow has caused the loss of sight.

How, then, shall we account for the blindness? There are only two possibilities; either it is a case of congenital amblyopia or of simulation. The patient might never have seen well with this eye and not have known it, because he never had any occasion to try the sight of his eyes separately. After the accident it was natural that he should try to find out whether or not the sight was injured; and then finding the eye almost blind, he would naturally and honestly believe the blindness was caused by the accident.

But, gentlemen, this is not a case of congenital amblyopia, but a case of simulated blindness. The man can see with his left eye, but affects blindness in order to obtain damages; and I am sure a few thousand dollars would effect an instantaneous cure of his blindness. I arrived at this conclusion by submitting the patient to a number of tests which I will repeat before you, and which I have no doubt will convince you of the correctness of my diagnosis. But before calling the patient back into this room let me explain the tests I propose to employ.

1. *The Test with a Convex Lens.*—Having ascertained that the right eye has normal vision and refraction, I shall put a convex lens of 4 diopters before this eye and let the patient read very small print (Sn. 0.5) with both eyes open. The 4 diopters lens makes the right eye myopic, so that it cannot read this fine print beyond ten inches. I shall hold the book at first at this distance from his eyes, and, of course, he will read it; then I shall gradually draw the book farther away, and if he continues reading without hesitation when the book is fifteen or twenty inches away, we know he is reading the print with the alleged blind eye. But if he should declare he cannot see the

print any more, we must not accept this statement at once, and conclude the left eye is really blind, for the malingerer may have received some pointer about this test and stop reading, although he may see the print perfectly plainly.

2. *The Prism Test.*—You know a prism deflects the rays of light towards its base. This action of prisms explains the fact that if while looking at a light you put a prism, base down, before your right eye, you see two lights, one above the other; for while your left eye receives the image of the flame upon the macula, the prism causes the image in the right eye to be cast upon a point of the retina below the macula, and gives you the impression of a second flame above the real one. For this test I select a prism of only five or six degrees, so that the two lights are near together and readily seen; and if our patient is very unsophisticated he may at once admit that he sees two lights and thus admit the simulation. But we shall be more certain to entrap him by modifying the prism test in the following manner: The left eye being covered, the prism, base down, is put before the right eye in such a manner that its edge coincides with the horizontal diameter of the pupil. This produces monocular diplopia, because the rays passing through the upper half of the pupil are collected to an image in the macula, and the rays passing through the prism and the lower half of the pupil are collected to a second image below the macula. As the left eye is covered the patient will not be afraid to admit the diplopia. While the patient is seeing double images, you remove, as if by accident, the shade or hand which covers the left eye, but at the very same moment push the prism before the right eye upward so that it covers the entire pupil, and ask the patient whether he still sees the two lights; as he still believes the glass makes him see double with the right eye, he will unhesitatingly give an affirmative answer, and thus convict himself; for in the second position the prism produces diplopia only if both eyes are seeing.

3. *The stereoscope test* is undoubtedly one of the neatest traps with which to catch impostors. The very simple stereoscope I show you here answers our purpose. It consists, as you see, of a shallow wooden box; the one side is open to be put against the forehead and the eyes, the opposite side is closed by two sphero-prismatic lenses, between which a partition is erected so that neither eye can see the object put in the slide before the other eye. The strong prisms, base out, deflect the picture presented to the right eye to the left side and

the picture before the left eye to the right side. When these pictures are very dissimilar, they appear transposed, as, for instance, the letters P N are seen as N P. Now, our patient, believing the letter P, seen on the right side, is the one he sees with the right eye, told me the other day, and most likely will tell you to-day, that he sees the P only, but as this is visible only to the left eye, his statement convicts him at once. But if we put in the stereoscope two letters like F and L, the vertical bars are blended, and the horizontal bars of the F and L are joined to the vertical line above and below to make an E, and our patient, seeing one letter only, unhesitatingly said he saw an E, and thus again proved he could see with the left eye as well as with his right eye.

4. *The Test with Colored Letters.*—Here is a series of red and green letters printed on a black background. If you put a red glass before your right eye and close your left eye you can see the red letters only; and that is what our patient would see with both eyes open if his left eye was really blind; but as he sees with the left eye he will read the green letters as well as the red ones.

If you have no printed color-letters, you can easily extemporize a test chart by writing on a white paper letters alternately with a lead-pencil and a red pencil. The red glass before the right eye blots out all the red letters for this eye, and the patient can read the black letters only if his left eye be really blind. If he reads all letters, you may be certain the blindness of the left eye is simulated.

Gentlemen, with the tests I have described, and which we shall now try on our patient, you cannot fail to expose the shrewdest mauler, provided you proceed with the necessary diplomacy. Success depends a great deal upon your action. Never during the examination show by mien or words that you suspect or doubt his statements; listen with apparently great interest to his story; drop occasionally a word of sympathy; in fact, gain his confidence, and the battle is already half won.

Laryngology.

CLONIC SPASM OF THE SOFT PALATE WITH TICKING SOUNDS IN THE EAR; EPICONDYLALGIA.¹

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY POLYCLINIC.

BY PROFESSOR M. BERNHARDT,

Professor of Nervous Diseases at the University of Berlin, Germany.

GENTLEMEN,—I have two cases to show you to-day that are interesting because of their comparative rarity. The first has a neurological interest because the groundwork upon which it develops and the nerve or nerves involved in the production of the spasm are uncertain. There is also an anatomical interest in the case, for as yet the nerves that supply the soft palate are not surely and definitely known, and here, as it has in so many other parts of the nervous system, there is hope that pathology will supply the defects in our knowledge that physiology and dissection have so far been unable to fill up.

Our patient is a woman of thirty-five years, who has had six children, is healthy looking, certainly not anaemic, and thinks herself that she is in excellent health except for her single set of symptoms. About seven weeks ago she noticed a spasm of her soft palate, which began suddenly one morning shortly after rising, and has continued ever since. There has never been any spasm of her face nor of any other part, and she has never had any such symptoms before. Her family history is good. There has never been any nervous or mental disease in the family, and she has five brothers and sisters, three of them older than herself, who are living and well. Her own children are perfectly healthy.

The only thing to be seen on examination is this clonic cramp of

¹ Reported by James J. Walsh, Ph.D., M.D.

the uvula and soft palate, so readily to be seen when I have her open her mouth. There is a constant up-and-down movement of these parts, which I have counted on several occasions and found it occurred from one hundred to a hundred and twenty times a minute, being pretty constantly between one hundred and ten and one hundred and twenty. You can easily imagine what an effort it would be to attempt to repeat such a movement voluntarily as many times as this, yet here it goes on without the slightest movement, nay, against the patient's will. She says that she does not know whether it continues during sleep or not.

You notice that her voice is not hoarse and that her vocalization generally is not disturbed. Examination with the laryngoscope shows that the vocal cords are not involved, and that all the functions of the muscles of the larynx remain undisturbed. Externally no muscles of the neck are disturbed except those from the hyoid bone to the tongue. It is evidently this involvement of the hyo-glossi that occasions the slight tremor of the tongue that is to be noted, the base of the tongue being affected by the clonic spasm of these muscles.

In connection with the spasm there is another very interesting phenomenon. Occasionally, not continuously, and, as often happens in such cases, especially liable not to be present when you wish to demonstrate it, there is a ticking sound in the ear. This is not merely subjective, but may be heard some feet away from the patient. In a quiet room I have heard it at a distance of more than a metre away. It resembles somewhat the ticking of a watch, and occurs, like the spasmodic contractions of the palate, more than one hundred times a minute. In fact, as far as I have been able to determine, the palatal spasms are synchronous with the sounds. Examination of the ear shows that there is no movement of the ear-drum accompanying the spasm.

In certain spasms in the region of the distribution of the facial nerve the stapedius muscle has been supposed by Erb to be involved, and in its movements, by setting up variations of pressure within the labyrinth, to give rise to the subjective sounds that are heard. In spasm in the region of the trigeminus distribution there has been noted not infrequently a synchronous movement of the tympanum, supposed to be due to spasmodic contractions and relaxations of the tensor tympani. This gives rise to a subjective sensation of sound. Luce points out that this sound is the same as any one may produce

by making a firm contraction of his muscles of mastication. In the same way, but through the stapedius muscle and its action on the labyrinth in this case, we are able to produce a subjective sound by a firm contraction of the muscles of expression.

Here we have no spasm in either the *facialis* or *trigeminus* regions, and, besides, the sound is not merely subjective, but objective. It resembles in character very much the sound produced by pulling two moist surfaces apart. It is, in other words, such a sound as might be expected if the Eustachian tube should collapse and then be pulled apart. This would be caused by the muscles that lie around the tube, especially at its pharyngeal end, sharing in the spasm of the other muscles of this region.

What are we to think, then, as to the origin of this symptom complex? With absolutely no other symptoms present, we think at once of hysteria, but have not the slightest ground for the hypothesis. She is a calm, restful-looking, healthy woman, who has never had any trouble with her nerves. She has had no sudden or powerful emotion lately, no serious cause for worry or alarm, and she has not run down in weight or strength. She has full sensibility of her palate and pharynx; she has no narrowing of her fields of vision, and her color fields are normal. She does not suffer from vasomotor troubles, has no anomalies of sweat secretion, and sensibility is normal. Under such circumstances hysteria would be a name perhaps to cloak our ignorance of the etiology, but would scarcely serve to disguise it effectually.

As this spasmotic contraction of the palate and pharynx has been reported to have occurred with facial spasm, there comes the question whether this is to be considered as a partial facial spasm or not, a so-called *tic convulsif* in the region of the *facialis* distribution, but limited to the soft palate and pharynx. These tics convulsifs in certain muscle groups in the neck and face are not so rare. Even where a number of muscles are involved, not all of them are affected with like intensity. The muscles that are most frequently and severely affected are the *orbiculi palpebrarum*, the *zygomatici*, and the *levatores labii superioris*, and after these the *platysmata*, the *digastrics*, the *corrugators*, and the *frontalis*. Curiously enough, the *orbicularis oris*, whose frequent use for a number of different and complicated movements might seem to make it especially liable to neurotic conditions, is not often or seriously affected. Isolated spasm of the

muscles of the lower part of the face are much rarer than of those of the upper part, but they occur, and lip and chin muscles, or the digastrics or platysmata, may be in a constant state of spasm, while other muscles remain free.

But as to thinking our case here an isolated facial group spasm, there is very little authority. The subject of the connection between soft-palate spasm and facial spasm has only interested neurologists in recent years. In 1876 Erb said that he had never seen facial spasm complicated by spasm of the soft palate, and that he could find no mention of it in the literature. In 1878, however, Leube published a case of double facial spasm, in which there was spasm also of the soft palate and uvula. There was an interesting addition to the symptoms in the fact that the salivary secretion had also been disturbed, excessive salivation having been complained of, showing that the salivary fibres of the nerve as well as its motor ones had been subject to irritation.

Later Schüssler published a very interesting and seemingly positive case in which the left half of the soft palate not only shared in the spasmotic contractions of the left side of the face, but when the facial tic convulsif was relieved by the stretching of the facial nerve the palate also shared in the paresis which followed. In one reported case at least of synchronous spasm of the face, the soft palate, and the uvula there was an accompanying anomaly of taste; salty and sour substances were less perceptible to the taste on the affected side than on the other.

Oppenheim, in the recent edition of his text-book on nervous diseases, reasserts his former opinion that in cases of "*pure*" facial spasm almost never does an accompanying spasm of the soft palate and pharynx occur. He considers that it is practically always due to some complication. Gowers, who holds a similar opinion, thinks that there is a coincidental association of the two afflictions in certain cases, but that they are not due to a single lesion of the same nerve-stem. I myself have never seen spasm of the soft palate with facial spasm, though I have faithfully looked for it now for years. We have, besides, none of the causes present to which at least the beginning of facial spasm, tic convulsif, is usually attributed, for ordinarily it is considered as reflex to some irritation of the peripheral sensory nerves. There are no specially bad teeth, there is no irritative condition of the conjunctiva, no history of a foreign body on the conjunctiva before the begin-

ning of the spasm, no contractile scar in whose meshes a peripheral sensory nerve might be imprisoned and irritated; there is no wisdom-tooth erupting; there seems to be no old collection of inflammatory exudate near a carious tooth to set up reflexes, no inflammatory condition of the Eustachian tube, and no history of the passage of a Eustachian catheter and the like.

There seems to be no reason to consider that we have to do with a partial facial spasm then. In the cases where it occurred it was considered that the source of irritation of the facial nerve was above the geniculate ganglion, whence through the Vidian nerve, the greater and lesser petrosal nerves, and through the otic and sphenopalatine ganglia it gave and received the branches that either directly or reflexly set up the spasm of the soft palate and pharynx. That only those fibres should be irritated which go to the palate and pharynx would be very surprising, and yet we have absolutely no sign of irritation in any other part of the distribution of the nerve.

If we dismiss the idea of partial facial spasm, then we are thrown back upon the hypothesis that we have an independent affection here, but we are quite as much in the dark as ever as to the nervous origin of the spasm. We do not know exactly what nerve or nerves are distributed over special parts of the palate and pharynx. The *facialis*, the *trigeminus*, and the *vagus* are all supposed to give fibres to this region. Of late we have learned to think that the *vagus* forms much more of the motor supply of these parts than we used to think. The motor supply of the larynx, that used to be attributed to the *accessorius*, has now been transferred to the *vagus*, which is thought to supply both motor and sensory filaments to these parts. We may have to do, then, with an irritative condition of certain fibres of the *vagus*. How limited the affection is in that case we may readily deduce from the perfectly normal functional condition of the remaining extensive *vagus* distribution. It is not impossible, however, that the *hypoglossus* is here also affected, since the *hyoglossi* are involved in the spasm, and there are some suspicious twitchings in the *thyrohyoid*. In a word, the present condition of our knowledge of the anatomy and physiology of these nerves and muscles is insufficient to enable us to come to any definite conclusion in the premises. These cases are always deserving of the most careful study, since pathology may help, as I have said, and as you are aware that it has often done in other cases, in the matter of deciding the nerve distribution to the parts.

As to treatment, there would seem to be nothing to do but improve the general condition of the patient in the hope that the functional disturbance of the peripheral nervous system will disappear with the improvement in systemic tone. Had we any points of local irritation from which the spasm might arise reflexly, of course we should turn our attention to these. Even though the painful affection is at a distance from the place of spasm, it will often be found that its cure is followed by the disappearance, or, at least, by great relief, of the spasmodic condition. Unfortunately we have not in this case any reason for thinking that the origin is a local sensory irritation.

For the cases of general facial spasm, tic convulsif, many patients prefer to have a paralyzed set of muscles on one side of their face to a set whose continual grimacing makes them extremely noticeable among their fellow-men. I should myself never advise a neurectomy, but there are patients who accept it and its consequences when explained to them very readily. Here we have no definite procedure of that kind to look to, and, besides, the spasmodic movements are concealed from view, and so do not react upon the patient's disposition.

My own best results in many of these spasmodic conditions were obtained from the bromides, even in cases where there was no question of epilepsy. In general, electrical treatment does good, too, and for the present we shall content ourselves with these.

As to the prognosis, it is not very favorable. The condition has not existed very long, and so would seem to hold out hope of cure, but it is so entirely independent of local irritation that this hope is only slight. The affection may disappear spontaneously, but will almost surely recur on some slight irritation.

Our second case to-day, gentlemen, is a characteristic example of an affection that in a mild form is much more common than used to be thought. Perhaps the people of past generations did not expect so much from the physicians as do those of ours, and so did not bring for treatment little afflictions that, while they give troublesome symptoms, have no alarming aspects about them. The understanding of little bothersome ailments is, however, one of the most satisfactory things in ordinary practice, and even though you may not be able to cure them, the consciousness that you thoroughly understand the condition, and the assurance you can give the patient as to its absolute harmlessness and want of significance as regards any nervous

or systemic condition, are of themselves most important considerations.

Our next patient, a woman of forty-two years, is a widow who has to work hard for a living at her occupation of ironing clothes. By working hard she says that she means working always twelve hours a day, and on Saturdays and before holidays, when ironed things are in demand, fifteen and more hours. Of late she has developed a pain here in the muscles of the forearm, which seems to begin about the elbow, passes downward towards the hand, and greatly hinders the use of her arm.

When we have her expose her elbow we find no sign of inflammatory disturbance in this region. There is no swelling demonstrable when we carefully compare the two arms, no reddening, nor any increase of temperature in the part. When we have her point out more exactly where the pain begins and is worst, we find that she locates it mainly just over the external epicondyle of the humerus, a secondary centre of discomfort being the head of the radius just below it. The pain is not sharp, lancinating in character, but dull and continuous, and either disappears or is much lessened when the arm is put at rest.

We evidently have to do with an affection here that I separated from other similar affections in the forearm some years ago, and to which, later, Feré, of Paris, gave the expressive Greek name of epicondylalgia. You may see, thus, of what very practical use your knowledge of Greek will be to you in your medical life. It will enable you to give proper Greek names to the new diseases you discover, and keep you from wounding the fine etymological sense so common to medical men, and that has of late been more than once sadly imposed upon by the aberrant nomenclature of the non-hellenists.

Epicondylalgia occurs especially on the right side, for the very good reason that, being an occupation neurosis, the right arm is oftenest overused. It is, for an analogous reason, an affection of men rather than of women. In some thirty-five cases of the disease I have had but five women. It occurs especially in people who work hard and use their forearm a great deal. I have seen it in plasterers, whose work of spreading plaster calls for use of the hand and arm continuously, in carpenters occupied much with the saw, the plane, and the hammer, and in locksmiths, seemingly from the use of the file.

Others have reported it especially in blacksmiths and in stone-cutters. These seem to be the trades most affected by it.

It occurs almost without exception after the age of thirty, most of my own cases having occurred between forty and fifty. The only one of my patients under thirty was a young woman of twenty years, who after receiving an injury to her arm continued to practise for hours each day on the violin, despite the inconvenience occasioned by the injury. Two other of my female patients, like our patient of to-day, were widows, and had to work hard under discouraging circumstances for a living. Another one, a woman of the better class, noticed her epicondylalgia first after having carried an umbrella for some time in wind and rain that required a special effort for its support. Her symptoms developed especially whenever afterwards she had to hold up an umbrella. The literature shows that such cases are not so rare, but usually they have not been so definitely studied as to give them their proper place in nosology.

I have said that it occurred most frequently on the right; where it has been noted on the left the patients were either left-handed or their history showed that they had, during the time immediately preceding the development of their symptoms, over-exerted their left hands. Twice I have seen it on both sides, though much more pronounced on the right.

The pathogenesis of the disease is evidently the over-exertion of certain muscles of the forearm, especially the extensors of the hand and fingers. These are not alone called into play in performing motions that are properly extensile in character, but act also when the flexors alone seemed to be in action giving the point of support that enables these to properly bring their force into play. An example that shows how important this counteraction of the extensors during flexion is, and one which all of you will readily recall, is the weakness of hand pressure, where there is palsy of the extensors from lead-poisoning, or from a compression neuritis.

There is an unusually large number of muscles that have their origin from the external epicondyle of the humerus, or from the head of the radius lying just below it. The supinator longus and brevis, the extensor carpi radialis longus and brevis, the extensor digitorum communis and carpi ulnaris, and the anconeus all are attached here. One can easily understand how their repeated and forcible contraction might lead to slight tearing of the periosteum covering the epicondyle, and, as the periosteum is rich in nerves, this would account

very well for the pain that occurs with use of the arm, and either stopping completely or becoming much more bearable when the arm is put at rest.

At times when trauma has played an etiological rôle in the disease, and when the affection has occurred on both sides, or when there has been a special history of exposure of the arm to cold, I have thought that there might be some inflammatory process in the periosteum. In the double-sided cases this would perhaps be of a rheumatic character. As all external symptoms of inflammation are lacking, and as often there is even no special painfulness to pressure in this region, whatever periostitis exists is very slight, and may be neglected.

Whether a corresponding affection might not exist of the internal condyle of the humerus, from which a similar bunch of muscles, the flexors of the arm, take their origin, I cannot say for certain. In two of the thirty-five cases that I have had under observation there did seem to be also some tenderness here over the inner condyle, but it is clear that the affection is much rarer than in the external condyle.

The condition may last a good while and take at least some weeks before it improves very much, though its prognosis is, on the whole, not unfavorable. While it continues the affection is a serious hindrance to work. A workman whose occupation demands much employment of his hands and fingers will not be able to go on with it. Not only manual labor is prevented, but in many cases also the more delicate manipulations required for writing or even playing the piano.

In the matter of treatment the first thing to be thought of, of course, is that the patients should give up, for a time at least, the occupation at which the affection has been acquired. A few days of complete rest do more good than months of supposed watchfulness not to permit over-exertion, though the occupation is continued. Only on a few occasions when there was considerable tenderness present, localized over the epicondyle, did I deem it advisable to apply leeches at this point. When used they accomplished the desired result of relieving the tenderness. Usually I advise a wet roller for some days, then repeated paintings with iodine, and finally the application of the anode of a medium strong galvanic current. The faradic electric brush has been of use when applied over the tender points, but must not be used so as to cause pain or produce sudden contractions of muscles or it will do harm rather than good.

Dermatology.

HERPES ZOSTER.

BY JOHN E. HAYS, A.M., M.D.,

Professor of Dermatology in the Hospital College of Medicine, etc., Louisville,
Kentucky.

THE disease called herpes zoster has for its common name "shingles." The word herpes comes from the Greek, which means to creep, a name given to the disease by the ancients because of its tendency to creep around the trunk and from one part of the body to another. The word zoster is also from the Greek, which means girdle. The common name shingles was derived from the Latin *cinctulum*, which means a girdle.

Herpes zoster is a disease which has been recognized from a very early period; it was known by the ancients. I think Hippocrates was the first to give anything like a detailed description of the affection. Some of the early writers speak of the disease as being one of a peculiar or mysterious nature, an idea which prevailed not only among the laity but also the profession; and the statement is made in some of the early works on medicine, which statement has been handed down to us, that if the disease extends all the way around the trunk it proves rapidly fatal. This idea is still held by the laity to a greater or less extent, and the patient will often be found very much alarmed if he notices upon examination that the disease involves both sides of the body and shows a tendency to extend entirely around the trunk. He fears that, when the girdle or circle is made complete, a fatal termination is the inevitable result. The affection is almost invariably unilateral, and if it extends around to the opposite side it does not necessarily follow that death will result.

The distinctive feature of the disease is a vesicle. This is also

characteristic of another skin disease, the ordinary eczema. The vesicles in herpes vary in size from the head of a pin to that of a pea. As a rule, these vesicles are gathered together in little groups or collections, and they follow the course of some of the cutaneous nerves. The vesicles are generally discreet, but sometimes they are so thickly set together that they coalesce so as to form large, irregularly shaped bullæ. If the patient is seen sufficiently early the vesicles will be found to contain a thin transparent fluid. Later this fluid changes in color and becomes opaque, which is due to the presence of leucocytes. A few days later the contents of the vesicles become darker, sometimes even becoming purplish, due to the presence of blood, which gets into the vesicles through the soft parts. If the case is seen before the vesicle forms, there will probably be found a red spot, which is spoken of by French writers as the congestive stage of the disease. That soon gives way, however, to a fully formed vesicle, which varies in size as already stated. These vesicles undergo certain changes, and after a few days' duration they dry up. The contents will disappear either by drying up, or the vesicle may rupture, and then follows a period which is known as the stage of desquamation.

Then comes the last stage, which is known as the macular stage, when there is simply a spot which is brown in color; this also fades away, and usually the skin assumes its normal color and appearance. In some cases the skin of the involved patch becomes either lighter or darker in color than the surrounding parts. In my experience the skin is usually lighter, especially in old people, after all the contents of the vesicle have passed away.

So in herpes zoster there are four stages,—viz., the congestive stage, the vesicular stage, the desquamative stage, and the macular stage; and finally the skin resumes its healthy aspects, with possibly a slight change in color, a little lighter or darker than the surrounding parts.

Before the appearance of any of these manifestations upon the skin the patient will frequently experience pain; if not actual pain, he will have some uneasy sensations, which will probably last for from one to four days. Pain sometimes in cases of intercostal herpes is so great as to lead the patient to believe that he has an attack of pleurisy. Pain in some cases is sufficient to even mislead the physician, unless a careful examination is made, and cause him to believe that he has a case of costal neuralgia or pleurodynia. In one case I

remember that a most excellent physician made the diagnosis of pleurodynia before appearance of the characteristic eruption. The pain of herpes is usually spoken of as burning and itching in its character. Sometimes the pain and itching will disappear upon appearance of the eruption; in other cases these sensations of itching and burning will continue throughout the eruptive stage and even after all traces of the eruption disappear, and particularly is this true in aged people. In young subjects the pain of herpes zoster is very slight, whereas in middle-aged and elderly people the pain is frequently severe and persistent. The reason for this is probably that the disease is really of an inflammatory nature, an inflammation of the nerve-trunks and also of the ganglia with which these sensory nerves are connected, resulting in inflammation and swelling, and in young subjects, by reason of the fact that the investing membrane of the nerve stretches when the effusion takes place, pressure is prevented to much greater extent than it would be in elderly subjects where the nerve membrane is not so elastic. I recently had a case in a baby where the mother noticed, in giving the child a bath, that there was an eruption on the right side along the track of the ileo-hypogastric nerve, to which my attention was directed. The baby had not been fretful during the night, and the mother did not suspect any trouble until it was stripped in the morning. All evidence of pain disappeared upon appearance of the eruption spoken of. That condition will frequently be observed in childhood, but in middle life and in the aged pain will persist during a number of weeks or even months after the eruption has disappeared. I have had patients with zoster affecting the trunk complain afterwards as if something was "binding" them.

The vesicles of herpes zoster differ from those of eczema in that the former rarely exhibit a tendency to rupture spontaneously; as a rule, they do not rupture, but gradually shrivel and dry up, the scale falls off, and a spot is left which after a time disappears.

This disease may occur upon any part of the skin which is supplied by sensory cutaneous nerves, especially those nerves which pass directly from the ganglia. The sensory nerves of the trunk pass from the ganglia, thence along the nerve-trunks up into the anterior and posterior branches, around the side of the chest as far forward as the median line in front.

The practical point to remember in the differentiation between this disease and eczema is that the vesicles of herpes are clustered

together in patches following the track of the sensory nerves, and although they itch like they do in eczema, they do not break down and discharge their contents. In eczema the vesicles are very evanescent; they readily break down, leaving an oozing surface, and nothing the physician can do will prevent rupture taking place; the patient may also have itching, but not the burning sensation which is present in herpes.

The life history of the vesicles of herpes is on an average twelve or fourteen days, beginning as a red spot, which soon gives place to a vesicle filled with fluid which changes in character as the days pass by; then the vesicle shrivels and dries up, which is known as the desquamative stage, after which the scales come off, leaving a more or less pigmented condition, known as the macular stage. This occupies about two weeks. If, however, the patient ruptures several of the vesicles, there is formed a superficial painful ulcer which may persist for several weeks, as it has exposed the terminal filaments of these sensory nerves; even if the nerves were not exposed, the pain would be considerable, for in this disease the intercostal nerves are inflamed, and there is a neuritis of the peripheral extremities of the nerves, which structures are brought in contact with the air, thus producing a great deal of pain. There is seldom any annoyance associated with the case except pain. Pain, however, is usually so severe in old people as to interfere with rest, and it becomes necessary in the management of these cases to give something to produce sleep.

The cause of this disease is more or less obscure, but it is supposed that the cause can be traced to one of several different things. It may be traumatic. If one of these sensory cutaneous branches has been injured by mechanical means, it may result in a crop of these vesicles along the course of the nerve. Then there are other causes, which are regarded as toxæmias. Sudden joy or grief may produce such an influence upon this nerve and ganglia as to produce a crop of herpes. Then, again, the same condition may be produced from the administration of arsenic, either large doses for a short time or small doses continued for a considerable length of time having been known to bring about this result. This fact has been brought prominently before the profession by Hutchinson, of London. Another set of causes are the miasmata. We find that malarial poisoning is the probable cause of many of these cases, especially in districts which are known to be malarial.

In making the diagnosis the physician must remember the predilection this disease has for certain parts of the body; when affecting the trunk it is known as herpes zoster, while when it occurs around the lips and about the face it is known as herpes labialis. It may extend around the lips and on the side of the face and cheek during the course of a pyrexial disease, or herpes may occur on the genitals, when it is known as herpes progenitalis,—all these forms presenting the same general characteristics. There are only three forms of herpes now regarded as typical, the zoster, labialis, and progenitalis. Fever blisters occurring upon the lips are usually painful for a few days, then dry up and disappear. Where herpes occur upon the genitals they are likely to be confounded with venereal disease. A patient came to me recently with a sore upon his penis. He had consulted an irregular physician, an advertising charlatan, who diagnosed the case as one of syphilis, and wanted the patient to pay him, I think, a hundred dollars before he would begin the treatment. The patient, not having the money, left the office of the quack and consulted a friend of mine, who referred him to me. In examining his penis I found he had simply a slight attack of herpes progenitalis. I advised him to do nothing but keep the parts clean. In a few days the vesicle disappeared, he went home, and has had no further trouble since.

As to treatment: To improve the constitutional condition of the patient the physician should use such remedies as may be indicated; cathartics are useful, many authors attributing the disease to a constipated condition of the bowels. As internal treatment I give a combination of the phosphide of zinc and belladonna, one-tenth grain each, every three hours. This is supposed to have the effect of shortening the attack and also of lessening the amount of pain. At night, if the pain should recur, to enable the patient to obtain the proper amount of sleep it is necessary to use some anodyne.

Local Treatment.—This is by far the most important, and the indications in the local management of these cases is to protect the vesicles from rupture and to allay the itching and burning sensation which is so often complained of. The most serviceable application in my hands has been the oil of peppermint, or a two-per-cent. solution of menthol in olive oil may be used. Many authors advise the use of applications such as the various dusting powders,—powdered boric acid, or calomel, or a combination of these, together with the

same amount of starch, keeping the spots covered with this powder, over which a layer of absorbent cotton is placed. Others, again, advise the use of different kinds of ointments, but ointments are not suitable, in my judgment, for local treatment of these cases. If the patient has broken the vesicles, and there are superficial ulcers, I think very often the condition may be benefited by using the lotion ordinarily known as the lead and laudanum lotion. A layer of absorbent cotton may be saturated with this lotion and laid over the painful ulcer, and will give a considerable amount of relief. After the ulcers have disappeared, if the pain still persists over the site of the disease, good may be done by painting the parts with a camphor and chloral mixture. This liquid applied over the painful parts does a great deal towards controlling the pain. The use of the galvanic current, probably from three to five milliampères, early in the attack, is said to lessen its severity and also to control the pain. This is a method of treatment that I have not yet employed, but it is to be thought of in connection with the disease. There are a great number of different things recommended as local remedies, but none will give such satisfaction as the oil of peppermint. It has been my custom for several years to apply the oil of peppermint by means of a camel's-hair brush. It eases the pain and the burning sensation, it also acts as a local anæsthetic, and I think the vesicles dry and the watery elements disappear more speedily under this application than anything else. A good many German authorities think well of ichthyol as a local application in this disease. Others think highly of painting the parts with collodion, washing the area carefully, and keeping the vesicles covered by a thin layer of collodion. But I think good reasons can be shown for adhering to the oil of peppermint after it has once been tried, and all the others I have mentioned.

INDEX TO VOLUME III.

(NINTH SERIES.)

A.

Abdomen, case of penetrating wound of the, 252.
Abdominal hemorrhage, case of, 251.
Abscess, case of pelvic, 265.
Anæmia among workers in brick-yards, frequency of, 143.
causes of, 140, 142.
hemorrhage as a cause of, 140.
loss of body fluids as a cause of, 140.
series of causes of, 141.
severe, connection between intestinal parasites and, 142.
tumors as a cause of, 141.
Anchylostomiasis and Graves's disease, 139.
Anchylostomum duodenale, description of, 144.
eggs of, 143.
results of, 143.
Aneurism as a cause of pulmonary hemorrhage, 168.
Aphasia, case of, 261.
Apoplexy during pregnancy, 255.
treatment of, 264.
usual sex for, 256.
Appendicitis, incipient, expectant treatment of, 16.
opiate treatment of, 15.
purgation in, 16.
stools in, 15.
treatment of, 15.
temperature in, 17.
Arteries, condition of, in Basedow's disease, 262.
Aspiration, danger of syncope in, 49.
method of performing, 48.
Atmosphere, dry, effect of, on the human body, 12.
saturation point of, 8.
August, description of hygrometer of, 9.

B.

Bang on tuberculosis among cattle, 2.
Basedow's disease, condition of arteries in, 262.
larval form of, 146.
pathognomonic sign of, 146.
prognosis of case of, 147.
Steilwag's symptom of, 145.
treatment of case of, 147.

Basedow's disease, triad of symptoms of, 145.
von Graefe's symptoms of, 146.
Bile, amount of, 154.
Birth palsy, symptoms of, 225.
Blindness and its detection, the simulation of, 272.
congenital amblyopia as a cause of, 273.
Bone lesions of hereditary syphilis in children, 224.
Brain, disturbed circulation and its effects on the, 107.
healthy, hygienic control of, 114.
tumor as a cause of optic neuritis, 210.
tumors, best diagnostic means of determining, 257.
choked disk in, 257.
Bright's disease as a cause of optic neuritis, 212.
Bromides in the treatment of clonic spasm of the soft palate, 281.
of ticking sounds in the ear, 281.
Bronchitis, chronic, symptoms of, 112.
Broncho-pneumonia, effect of, on the brain, 108.
Butter as a medium of infection by tuberculosis, 2.

C.

Cancer of the oesophagus, communication of, to the lung, 23.
complications of, 23.
danger of cough in, 88.
operation for, 87.
prognosis of, 23.
treatment of, 24.
Cataract, results of removal of crystalline lens for, 100.
Cattle, consequences of tuberculosis among, 1.
Cerebral circulation, illustrations of disorders of the, 108.
Chancres, case of, 117.
Children, methods of administering mercurial baths to, 230.
objections to mercurial injections in, 230.
Chlorosis, diagnosis of, 142.
Chorea, cases of, 73, 76, 77.
dose of antipyrrin in the treatment of, 74.
precaution in using antipyrrin in, 75.
table of nineteen cases of, 73.
treatment of, by antipyrrin, 73.
by caffein citrate, 75.

Chorea, treatment of, by chloral, 75.
 by diet and Fowler's solution, 74.
 by phenacetine, 75.
 by rest in bed, 74.
 by syrup of the iodide of iron, 74.

Circulation, disordered, differentiation of, from primary brain disturbance, 114.
 disturbed, and its effects on the brain, 107.
 case showing effects of, on the brain, 109.
 relief of, by venesection, 112.

Circulatory phenomena during pregnancy, 262.

Clamp method for closing lateral lesions of veins, 241.
 of haemostasis, objections to, 243.

Clonic spasm of the soft palate, 276.

Complications of croupous pneumonia, 184.

Condyloma latum, prescription for, 231.

Cow's milk, centrifugation and conversion of, 4.
 dilution of, 3.
 new method of heating, 6.
 sterilization of, 6.
 uses of, 2.

Croupous pneumonia, the complications of, 184.

Cyclic vomiting in children, 127.
 auto-intoxication as a cause of, 134.
 cases of, 127, 128, 129.
 cause of vomiting in, 134.
 conditions causing attack of, 137.
 description of typical attack of, 131.
 first noticeable symptom of, 130.
 food in cases of, 137.
 gastro-intestinal inflammation in, 136.
 Holt on, 133.
 important facts in the history of cases of, 134.
 literature on, 133.
 marked vomiting in, 131.
 microscopic appearance of appendix in, 130.
 nature and cause of, 132.
 neurotic element in, 137.
 Pepper on, 133.
 post-mortem notes on case of, 135.
 removal of appendix in, 130.
 Rotch on, 133.
 temperature in, 131.
 tendency of cases of, 137.
 treatment of, 138.
 urine in case of, 137.

Cystitis, gonorrhœal, urine in, 35.

D.

Dairy techniques, some improvements in, 1.

Diabetes, causes of, 158.
 classes of, 158.
 clinical forms of, 159.
 complication of, 158.

Diabetic coma and its treatment, 156.
 case of, 156.
 diagnosis of, 158.
 evolution of, three classes of, 159.
 Gerhardt's reaction in, 157.
 heart in, 157.
 intoxication as a cause of, 159.
 nervous symptoms in, 157.
 prescriptions for the relief of, 161.
 prognosis of, 160.
 serious complication of, 156.
 theories as to the origin of, 159.
 treatment of, 160.
 by antipyrine, 156.
 urine in, 156.

Diagnosis of cancer of the œsophagus, 21.
 of malignant neoplasm of the vertebræ, 19.
 of neurasthenia, 195.
 of stricture of the œsophagus, 22.

Diarrhoea, infantile, effect of, on the brain, 108.

Diet in neurasthenia, 196.

Differentiation of neurasthenia and hypochondria, 195.
 and hysteria, 195.
 and melancholia, 195.

Diphtheria, antitoxin in the treatment of, 90.
 cause of, 89.
 differentiation of, from membranous croup, 89.
 local treatment of, 92.
 method of employing antitoxin in, 91.
 remarks upon the treatment of, 93.
 syringing in the treatment of, 93.
 treatment of, by antiseptics, 92.
 by drugs, 92.

Diseases of the endocardium, Pepper on, 180.

Disturbed circulation, clinical aspects of case of, 111.
 prognosis of, 111.
 urine in case of, 110.

Drugs in neurasthenia, 198.

E.

Ear, ticking sounds in the, 276.

Eczema and herpes zoster, differentiation of, 287.
 characteristics of, 288.

Emboli as causes of disturbance of brain function, 179.
 of hemorrhagic infarction of the lung, 178.
 consequences of, 178.
 mitral disease as a complication of, 179.
 most frequent seats of, 179.
 principal origin of, 178.

Embolism, bearing of, on valvular heart-disease, 177.
 in heart-lesions, cases of, 181, 182, 183.
 effects of, 181.
 of a branch of the renal artery, case of, 182.
 of the mesenteric artery, case of, 183.

Endocarditis, symptoms of, 185.

Endocardium, Pepper on diseases of the, 180.

Enlargement of the heart, case of, 258.

Enteric fever, blood-pressure in, 113.

Epicondylalgia, case of, 282.
most usual age for, 283.
occupations most frequently the cause of, 282.
pathology of, 283.
prognosis of, 284.
sex usually affected by, 282.
symptoms of, 282.
traumatism as a cause of, 284.
treatment of, 284.
usual side for occurrence of, 282.

Erb on facial spasm, 279.

Etiology of neurasthenia, 193.

Evaporator, description of an, 11.
in the living room, therapeutic value of, 7.

F.

Facial spasm, Erb on, 279.

Fat-milk, preparation of, 4.
results of using, 5.

Favus in mice, 105.
of the nail, after-treatment in operation for, 106.
description of, 105.
differentiation of, 105.
Glueck on, 105.
location of, 106.
treatment of, 105.
by avulsion, 106.

Fever, typhoid, case of, in a child, 173.

Fistulae, tuberculosis as a cause of, 221.

Fracture of the patella, cause of, 52.
difficulties in the treatment of, 57.
examples of old, 56.
factors delaying union in, 53.
preventing apposition of fragments in, 53.
function after union of, 55.
hemorrhagic effusion in, 54.
joint complications of, 52.
muscular spasm in, 54.
peculiarity of, 52.
symptoms of old, 56.
treatment of, 58.
after apposition of fragments, 55.
von Bergman's, 57.

G.

Gangrene following embolism, 180.
mitral stenosis, case of, 180.

Gastric juice, amount of, 154.

Gerhardt's advice concerning heart-murmurs, 178.

Glasier's table of relative humidity, 9.

Glueck on favus of the nail, 105.
on haemostasis, 244.

Gowers on spasm of the soft palate, 279.

Graves's disease, effect of, on the circulation, 114.
larval form of, 146.
pathognomonic sign of, 146.
prognosis of case of, 147.

Stelwag's symptom of, 145.
treatment of case of, 147.
triad of symptoms of, 145.
von Graefe's symptom of, 146.

H.

Haemostasis, Glueck on, 244.
Heinicke on, 245.
Horoch on, 244.
Jasinowsky on, 245.
Niebergall on the clamp method of, 241.
objections to the clamp method of, 243.
Péan's method of, 241.
Simpson's method of, 241.
suture as a means of, 243.

Heart, case of enlargement of the, 258.
Gibson on the treatment of chronic affections of the, 177.
physical sign of hypertrophy of the, 263.
prognosis in valvular lesions of the, 177.

Heinicke on haemostasis, 245.

Hemiplegia, case of, 256.

Hemorrhage, case of abdominal, 251.
cases of acute traumatic abdominal, 248.
pulmonary, aneurism as a cause of, 168.
death from, 163.
diagnosis of, 167.
due to aneurism, symptoms of, 168.
origin of blood in, 167.
treatment of, by ice, 163.
by salt, 164.
hypodermically, 166.
secondary, cause of, 236.

Hemorrhoids, frequency of, 217.
in the Indian, rarity of, 218.
in the negro, rarity of, 217.

Heredity as a cause of neurasthenia, 193.

Herpes progenitalis, case of, 289.
three forms of, 289.
zoster and eczema, differentiation of, 287.
a vesicle as the characteristic symptom of, 285.
cause of, 288.
diagnosis of, 289.
differentiation of, 287.
distinctive feature of, 285.
four stages of, 286.
pain in, 286.
shingles a common name for, 285.
traumatism as a cause of, 288.
usual location for, 287.

High myopia, cause of, 97.
operative treatment of, 97.
results of, 100.

Horoch on haemostasis, 244.

Human body, effect of dry atmosphere on the, 12.
percentage of water in, 7.

Humidity, symptoms of excess of, 13.

Hutchison triad of symptoms of hereditary syphilis, 233.

Hygrometer of August, description of, 9.

Hypertrophy of the heart, physical sign of, 263.

Hypocondria and neurasthenia, differentiation of, 195.

Hysteria and neurasthenia, differentiation of, 195.

I.

Infarct, kinds of, 178.

Infection by tuberculosis, butter as a medium of, 2.

Iodides in hereditary syphilis, 233.

J.

Jasnowsky on haemostasis, 245.

Joint-fractures, treatment of, 51.

K.

Kidney and spleen, case of rupture of, 253.
case of rupture of, 252.
tuberculosis of the, origin of, 37.
usual location of, 36.

Kidneys in pregnancy, 259.

L.

Laryngeal obstruction, antitoxin in the treatment of, 93.
intubation in the treatment of, 94.
method of removing tubes in, 95.
O'Dwyer's tubes in the treatment of, 94.
symptoms of, 93.
technique of intubation in, 95.
usual mistakes in performing intubation for, 95.

Lateral clamping of injured veins, Porter and Pirogoff on the use of, 240.
ligature as a means of haemostasis in vein injuries, 238.

Blasius on the objections to, 238.
Braun on the use of, 239.
objections to, 240.
von Wattmann on the use of, 238.
openings in the veins, methods of treating, 236.

Lead-poisoning as a cause of optic neuritis, 211.
most important signs of, 214.
usual method of causing, 215.

Liver, carcinoma of the, case of, 174.
condition of bowels in, 176.
treatment of, 175.

case of stab-wound of the, 250.

Locomotor ataxia, early sign of, 213.
optic-nerve degeneration in, 210.

Lymphoma of the neck, case of, 234.
operation on, 234.

M.

Malignant neoplasm of the vertebral, case of, 18.
diagnosis of, 19.
pain in, 19.
symptoms of, 19.
treatment of, 20.

Massage in neurasthenia, 197.

McDowell's operation in gynaecology, 266.

Measles, diagnosis of, 170.
rarity of second attacks of, 171.

Melancholia and neurasthenia, differentiation of, 195.

Meningitis as a complication of pneumonia, 185.

Mercurial baths, method of administering, to children, 230.
injections in children, objections to, 230.
intoxication, albuminuria in, 117.

Mercury in syphilis, Guns's opinion on, 116.

Methods of haemostasis, objections to clamp, 243.
Simpson's and Péan's, 241.

Milk, centrifugation of, 4.
conversion of, 4.
cow's, uses of, 2.
dilution of, 3.
human, taste of, 6.
in neurasthenia, 196.
new method of heating, 6.
preparation of fat-, 4.
results of using fat-, 5.
Schlüter's investigations in, 3.
sterilization of, 6.
von Noorden on the uses of, 4.

Multiple injuries, case of, 251.

Myopia, cases of, suitable for operation, 101.
cause of, 97.
contra-indication to operation for, 102.
effect of a concave lens in cases of, 99.
high, 97.
indications for operating on cases of, 102.
method of operating for, 102.
points demonstrating when both eyes should be operated on for, 104.
preferable age for operating on cases of, 102.
removal of lens for, 97.
results of operation for, 98.
technique of operating for, 103.
treatment of, operative, 97.

N.

Nail, favus of the, after-treatment in operation for, 106.
description and differentiation of, 105.
Glueck on, 105.
location and treatment of, 106.
treatment of, 105.

Nephritis, gonorrhœa as a cause of, 34.
mercury as a cause of, 116.
prognosis of different kinds of, 117.
Rovsing's suggestions in, 39.
syphilis as a cause of, 116.
Tait's suggestions in, 39.
tuberculous, treatment of, by exploratory incision, 41.
by nephrectomy, 42.

Nerve, optic, degeneration of, in locomotor ataxia, 210.
importance of, 209.

Nervous symptoms following traumatism, 200.

Neurasthenia and hypocondria, differentiation of, 195.
and hysteria, differentiation of, 195.
and melancholia, differentiation of, 195.

Neurasthenia, causes predisposing to, 193.
 diagnosis of, 195.
 diet in, 196.
 differentiation of, 195.
 drugs in, 198.
 etiology of, 193.
 heredity as a cause of, 193.
 massage in, 197.
 milk in, 196.
 most important cause of, 193.
 overwork as a cause of, 193.
 physiology of, 193.
 principles governing treatment of, 196.
 prognosis of, 196.
 rest-treatment of, 198.
 symptoms of, 194.
 traumatism as a cause of, 194.
 treatment of the worst cases of, 196.
 usual age for, 193.
 Neuritic paraplegia with atrophy, treatment of, 70.
 Neurosis, traumatic, and a question of damages, 199.
 traumatism as a cause of, 201.
 Niebergall on the clamp method of haemostasis, 241.
 Noorden, Von, on the uses of milk, 4.

O.

Objections to clamp method of haemostasis, 243.
 Objective symptoms, nervous, following traumatism, 202.
 Oesophageal stricture, 22, 80.
 Oesophagus, cancer of the, cases of, 20, 86.
 cough in, danger of, 88.
 diagnosis of, 21.
 operation for, 87.
 prognosis of, 23.
 treatment of, 24.
 by oesophagotomy, 86.
 stricture of the, 22, 80.
 impermeable, case of, 81.
 passage of liquids through, 81.
 permeable, treatment of, 84.
 rectal feeding in, 83.
 regurgitation in, 81.
 treatment of, by gastrostomy, 84.
 by retrograde dilatation, 84.
 Gussenbauer's, 85.
 oesophagoscope in, 85.
 Soini's, 83.
 surgical, 78.
 von Hacker's, 82.
 Operation of ovariotomy, description of, 270.
 drainage in, 270.
 stimulants after, 271.
 Ophthalmoscope in the diagnosis of brain tumor, 257.
 Opium in the treatment of pain in appendicitis, 17.
 Oppenheim on spasm of the soft palate and pharynx, 279.
 Optic atrophy, case of, 213.
 nerve degeneration in locomotor ataxia, 210.
 importance of degeneration of, 209.
 nerves, anatomy and pathology of, 210.
 neuritis as a symptom of brain tumor, 211.

Optic atrophy as an early symptom in nervous diseases, 209.
 brain tumor as a cause of, 210, 211.
 Bright's disease as a cause of, 212.
 causes of, 210.
 forms of poisoning causing, 211.
 lead-poisoning as a cause of, 211.
 symptoms of, 212.
 syphilis as a cause of, 211.
 Ovariotomy, 265.
 description of operation of, 270.
 drainage in, 270.
 stimulants after, 271.
 technique of performing, 267.
 Overwork as a cause of neurasthenia, 193.

P.

Pain in appendicitis, opium for, 17.
 treatment of, 17.
 Palate, clonic spasm of the soft, 276.
 spasmodic contraction of the, 279.
 Palsy, symptoms of, 258.
 Pancreatic juice, amount of, 154.
 Paracentesis, method of performing, 48.
 Patella, fractures of the, cause of, 52.
 complications of, 50.
 dressing of, 51.
 factors delaying union in, 53.
 preventing apposition of fragments in, 53.
 hemorrhagic effusion in, 54.
 joint complications of, 52.
 peculiarity of, 52.
 reason for bad results in treating, 50.
 rules for treating, 50.
 treatment of recent and old fractures of the, 50.
 Pathology of epicondylalgia, 283.
 Péan's method of haemostasis, 241.
 Pelvic abscess, case of, 265.
 Penetrating wound of the abdomen, case of, 249.
 Pericarditis, case of, 232.
 differentiation of, from pleurisy, 184.
 symptoms of, 232.
 with effusion, symptoms of, 185.
 Physiology of neurasthenia, 193.
 Pleurisy as a complication of pneumonia, 184.
 differentiation of, from pericarditis, 184.
 with effusion, advantages of exploratory puncture in, 45.
 aspiration for, 47.
 best aspirator for use in, 47.
 causes of, 43.
 classical signs of, 44.
 dangers of exploratory puncture in, 45.
 diet in, 46.
 disadvantages of exploratory puncture in, 45.
 heart-failure in, 46.
 iron in, 46.
 paracentesis in, 47.
 physical diagnosis of, 44.
 signs of, 45.
 rest in, 46.
 signs of, 44.

Pleurisy with effusion, treatment of, by aspiration, 48.
by drugs, 45.

Pneumonia and phthisis, differentiation of, 190.
and typhoid fever, differentiation of, 191.
case of, 253.
cases of, most likely to be overlooked, 190.
characteristic sputum in, 187.
clinical varieties of, 186.
complicating tuberculosis, case of, 186.
complications of croupous, 184.
conditions modifying the prognosis of, 189.
difference in, and typhoid fever, 186.
difficulties of diagnosis of, in children, 190.
ease of diagnosis of, 190.
hospital treatment of, compared with house treatment, 189.
malarial type of, 186.
malignant, diagnosis of, 186.
methods of termination, 189.
migratory, description of, 188.
prognosis of, 188.
mortality in, 189.
prognosis of, in drunkards, 187.
sequelæ of, 189.
treatment of, 188.
typhoid type of, 187.

Poisoning, forms of, causing optic neuritis, 211.

Posterior sclerosis, bladder weakness in, 213.

Predisposing causes of neurasthenia, 193.

Pregnancy, albuminuria in, 259.
apoplexy during, 255.
treatment of, 264.

circulatory phenomena during, 262.
kidney of, 259.
urine in, 259.

Principles governing the treatment of neurasthenia, 196.

Prognosis of clonic spasm of the soft palate, 281.
of epicondylalgia, 284.
of neurasthenia, 196.
of syphilitic epiphysitis, 227.
of ticking sounds in the ear, 281.

Pseudoparalysis syphilitis of Parrot, 226.
pathological changes in, 226.

Pseudotubes, symptoms of, 214.

Pulmonary hemorrhage, aneurism as a cause of, 168.
cases of, 163, 164.
death from, 163.
diagnosis of, 167.
due to aneurism, symptoms of, 168.
hypodermic treatment of, 166.
ice in the treatment of, 163.
origin of, 164.
of blood in, 167.
relation of, to extent of lung involvement, 165.
salt in the treatment of, 164.

R.

Rachitis as a result of hereditary syphilis, 221.

Rectal diseases, general remarks upon, 216.

Renal syphilis, Mauriac's division of cases of, 120.

Respiration, effect of, on the fontanelle, 108.

Rest-treatment of neurasthenia, 198.

Rubeola, differentiation of, from scarlet fever 172.
Hénoch's opinion on, 171.
rash in, 171.
resemblance of, to measles, 170.
treatment of, 173.

Rules governing the suturing of large veins, 247.

Rupture of the kidney, case of, 252.

S.

Scarlet fever, differentiation of rubeola from, 172.

Schüssler on spasmodic contractions of the soft palate, 279.

Schütz's investigations of milk, 3.

Secondary hemorrhage, cause of, 236.

Shingles as a common name for herpes zoster, 285.

Simpson's method of haemostasis, 241.

Simulated blindness, case of, 273.
colored-letter test for, 275.
prism test for, 274.
stereoscope test for, 274.
testing for, with a convex lens, 273.

Soft palate, clonic spasm of the, 276.

Spasm, clonic, of the soft palate, 276.

Spleen and kidney, case of rupture of, 253.

Sterilization of milk, 6.

Stomach, acute formation of gas in the, 149.
case of, 151.
causes of, 149.
commonest kind of, 149.
danger of, 151.
potassium test in, 150.
salol test in, 150.
symptoms of, 151.
treatment of case of, 151.
uncommon types of, 150.
vomiting in, 150.

Chemic respiration within the, 153.

distended, case of, 151.
explanation of, 153.
treatment of, 152.

mechanic respiration within the, 153.

Stricture of the oesophagus, diagnosis of, 22, 79.
first symptoms of, 79.
gastrostomy for, 84.
regurgitation in, 81.
retrograde dilatation for, 84.

Subjective nervous symptoms following traumatism, 206.

Suture as a means of haemostasis, 243.
of a vein, case of, 243.

Sutures, various kinds of, 246.

Suturing wounds of large veins, rules governing, 247.

Symptoms, Hutchison triad of, of hereditary syphilis, 233.
nervous, following traumatism, 202.
of birth-palsy, 225.
of epicondylalgia, 262.
of hereditary syphilis, 213.

Symptoms of malignant neoplasm of the vertebral column. 19.
 of neurasthenia, 194.
 of optic neuritis, 212.
 of palsy, 258.
 of pericarditis, 232.
 of pseudotabes, 214.
 of syphilitic epiphysitis, 225.
 osteitis, 225.
 of tuberculosis, 25.
Syncope, case of, 113.
 cause of, 114.
Syphilis as a cause of optic neuritis, 211.
 hereditary, diet in cases of, 123.
 drugs in, 124.
 forms of, 123.
 gumma in, 124.
 Hutchison triad of symptoms of, 233.
 in infancy, 125.
 iodide treatment of, 123.
 mercurial treatment of, 123.
 prescriptions for, 125.
 rachitis as a result of, 231.
 specific treatment of, 122.
 symptoms of, 213.
 teeth in, 233.
 in the negro, frequency of, 220.
 of the kidneys, symptoms of, 122.
 urine in, 122.
 tardy kidney complications of, 117.
Syphilitic epiphysitis, case of, 227.
 symptoms of, 225.
Nephritis, albumen in the urine of cases of, 118.
 anasarca in, 118.
 autopsy in case of, 119.
 causes of death from, 121.
 description of, 117.
 iodides in, 118.
 loss of weight in, 118.
 mercury in, 118.
 pathological anatomy of, 121.
 peculiarity of, 121.
 Perroud on case of, 120.
 prognosis of, 227.
 symptoms of, 118.
 treatment of, 228.
 usual time for development of, 121.
 Wickham on cases of, 120.
Osteitis, symptoms of, 225.

T.

Tabes dorsalis, contra-indications to exercises in, 72.
 to the use of strychnine in, 62.
 drugs for the relief of pains in, 65.
 electricity in, 67.
 exercise in, 67.
 four movements in the treatment of, 69.
 frequency of bone-fracture in, 66.
 gastric and intestinal crisis in, 65.
 heat for the relief of pains in, 65.
 illustration of movement therapy in, 71.
 incoordinations in, 66.
 iodides in the treatment of, 61.
 mercury in the treatment of, 61.

Tabes dorsalis, most prominent etiological factor in, 63.
 movement therapy in, 66.
 phosphate and phosphoric acid in, 64.
 preataxic stage of, 72.
 Romberg symptom of, 67.
 sea-bathing in, 63.
 strychnine in the treatment of, 61.
 three methods of compensating for muscle weakness, 70.
 treatment of, 60.
 bedridden patients with, 60.
 by baths and massage, 62.
 by exercise, 69.
 by movement, 68.
 by rest and diet, 63.
 by silver and ergotin, 64.
 by stretching the cord and nerve trunks, 64.
 by suspension, 64.
 incoordination of hands in, 72.
 pains in, 64.
 perforating ulcers in, 66.
 trophic disturbances in, 65.
 two classes of patients suffering from, 67.

Tachycardia, case of, 144.

Tea, effects of, on the nervous system, 115.

Technique of clamping a wounded vein, 241.
 of haemostasis in operative lesions of large veins, 234.
 of performing ovariotomy, 267.

Ticking sounds in the ear, 276.

Tobacco, effects of, on the nervous system, 114.

Traumatic neurosis, history of case of, 208.

Traumatism as a cause of epicondylalgia, 284.
 of neurasthenia, 194.
 of neurosis, 201.
 of tuberculous nephritis, 38.

Treatment, Basch's, of tuberculous nephritis, 41.
 Gussenbauer's, of stricture of the oesophagus, 85.
 hypodermic, of pulmonary hemorrhage, 166.
 methods of, of stricture of the oesophagus, 85.
 of apoplexy during pregnancy, 264.
 of bedridden patients with tabes dorsalis, 69.
 of cancer of the oesophagus, 24.
 by oesophagotomy, 86.
 of clonic spasms of the soft palate, 281.
 of diabetic coma, 160.
 of diphtheria, antitoxin in the, 90.
 by drugs and antiseptics, 92.
 locally, 92.
 remarks upon the, 89.
 of favus of the nail by avulsion, 106.
 of fractures of the patella after apposition of fragments, 55.
 recent and old, 50.
 of herpes zoster, constitutionally, 289.
 locally, 289.
 of incoordination in tabes dorsalis, 68.
 of hands in tabes dorsalis, 72.
 of joint-fractures, 51.

Treatment of malignant neoplasm of the vertebral column, 20.
 of neurasthenia, severe cases of, 196.
 of neurotic paraplegia with atrophy, 70.
 of pain in appendicitis, 17.
 of perforating ulcers in tabes dorsalis, 66.
 of permeable strictures of the oesophagus, 84.
 of pleurisy with effusion by aspiration, 48.
 by diet, iron, and rest, 46.
 by drugs, 45.
 by paracentesis, 47.
 of pulmonary hemorrhage by ice, 163.
 by salt, 164.
 hypodermically, 166.
 of rubella, 173.
 of stricture of the oesophagus by the oesophagoscope, 85.
 of syphilitic epiphysitis, 228.
 nephritis by mercury and the iodides, 119.
 of tabes dorsalis by exercise, 69.
 by mercury, 61.
 by movements, 68.
 of ticking sounds in the ear, 281.
 of triplegia, 70.
 of tuberculosis, 25.
 of tuberculous nephritis, 40.
 of typhoid fever in a child, 174.
 of uremia by hot-air baths and pilocarpine, 113.
 operative, of high myopia, 97.
 Socin's, of stricture of the oesophagus, 83.
 surgical, of tuberculous nephritis, 33.
 von Hacker's, of stricture of the oesophagus, 82.

Triplegia, treatment of case of, 70.
 Tuberous bacilli, development of, in the human body, 37.

Tuberculosis among cattle, Bang on, 2.
 consequences of, 1.
 butter as a medium of infection by, 2.
 climate in, 31.
 cod-liver oil in, 28.
 diet in, 26.
 fever in, 30.
 food in, 28.
 haemoptysis in, 31.
 liquids and desserts in, 27.
 location of, 222.
 of the buttock, symptoms of, 219.
 treatment of, 220.
 of the kidney, origin of, 37.
 usual location of, 36.
 overfeeding in, 29.
 remedies in, 25.
 sanatoria for the treatment of, 31.
 symptoms of, 25.
 temperature in, 29.
 treatment of, 25.
 usual dietary errors in, 26.
 weight of patients in, 27.

Tuberculous nephritis, Basch's treatment of, 41.
 exploratory incisions in, 41.
 nephrectomy for, 42.
 surgical treatment of, 33.
 traumatism as a cause of, 38.
 treatment of, 40.
 urine in, 33.

Tumor in submaxillary region, case of, 234.

Tumors, brain, best diagnostic means of determining, 257.
 choked disk in, 257.
 ophthalmoscope in the diagnosis of, 257.
 first evidence of existence of, 257.

Typhoid fever in a child, case of, 173.
 danger of dissemination of, 174.
 diet in, 174.
 symptoms of, 173.
 treatment of, 174.

U.

Uræmia, blood-pressure in, 112.
 treatment of, by hot-air bath, 113.
 by pilocarpine, 113.

Ureters, catheterization of, 39.

Urine in gonorrhœal cystitis, 35.
 in pregnancy, 259.
 in tuberculous nephritis, 33,

Uvula, clonic spasm of the, 277.

V.

Vein, case of a suture of a, 243.
 technique of clamping a wounded, 241.

Veins, compression as a means of haemostasis in the, 237.
 methods of treating lateral openings in the, 236.
 rules governing suturing wounds of large, 247.

Vomiting, cyclic, in children, description of typical attack of, 131.
 first noticeable symptoms in, 130.
 Holt on, 133.
 literature of, 133.
 marked vomiting in, 131.
 microscopic appearance of appendix in, 130.
 nature and cause of, 132.
 removal of appendix in, 130.
 temperature in, 131.

W.

Water, percentage of, in human body, 7.
 quantity of, in each cubic foot of air, 8.

Wound of the abdomen, case of penetrating, 252.

Wounds of large veins, rules governing the suturing of, 247.

UNIVERSITY OF MICHIGAN

